A dissertation on

EVALUATION OF CAUSES OF ACUTE PANCREATITIS – A RETROSPECTIVE STUDY

Dissertation submitted to

THE TAMIL NADU DrM.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

With partial fulfilment of the regulations required

for the award of degree of

M.S. GENERAL SURGERY

BRANCH- I

COIMBATORE MEDICAL COLLEGE,

COIMBATORE

MAY 2018

DECLARATION

I solemnly declare that the dissertation titled " **EVALUATION OF CAUSES OF ACUTE PANCREATITIS – A RETROSPECTIVE STUDY"** was done by me from 2016 onwards under the guidance and supervision of **PROF. DR. D.N. RENGANATHAN , M.S**

This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical University towards the partial fulfillment of the requirement for the award of M.S Degree in General Surgery (Branch I).

PLACE: DR. BALAJID.J.

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CERTIFICATE

Certified that this is the bonafide dissertation done by **DR.BALAJID.J.** and submitted in partial fulfillment of the requirement for the Degree of M.S. General Surgery, Branch I of the Tamilnadu Dr. M.G.R. Medical University , Chennai.

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Dissertation Topic : A STUDY ON EVALUATION AND CAUSES OF ACUTE PANCREATITIS - A RETROSPECTIVE STUDY

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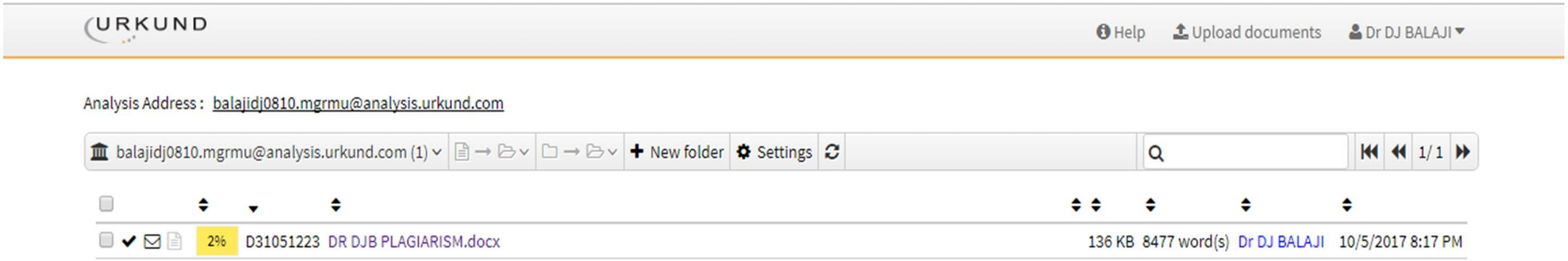
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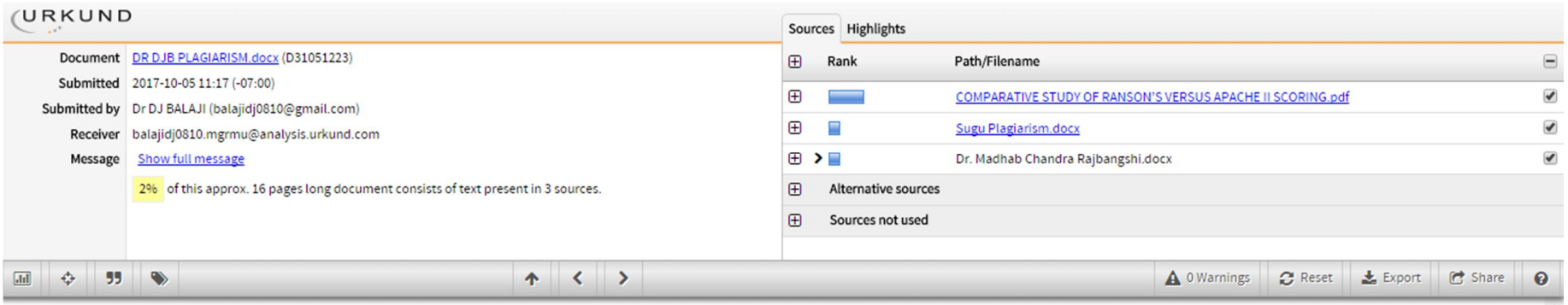

REVIEW OF LITERATURE HISTORICAL REVIEW : • In Greek Pan means "all" and Kreas means "Flesh" and it was described first by Herophilus Chalcedon. • Rufees and Ephesees – named the pancreas. • Wirsung – described the main pancreatic duct. • Santorini – illustrated the accessory duct bearing his name. • Ringor de Graaf – gave the first reference to pancreatic lithiasis in 1664. • Reiddle – described chronic pancreatitis. • Kini – reported the first case of pancreatic calculi in India. • Elizabeth and Stephen – reported 9 cases of pancreatic calculi from vellore. • Chuttani and Anand – reported 32 cases of pancreatitis from North India. • Brocks and Gifferd – performed the first human homotransplant in 1959 , using fragmented pancreatic tissue. • Lillehei and collegues – performed the first human pancreatic whole organ transplant in 1967. • Ballinger and Lacy – popularized the concept of islet cell transplantation in 1972.

INTRODUCTION Acute pancreatitis has become a common disease among the new world population due to the increase in the socio-economic factors affecting the general population which has led to an increase in risk factors associated with the onset of acute pancreatitis. Acute pancreatitis has become a formidable cause of mortality and morbidity in recent times due to its sheer clinical features and debilitating symptoms. The increased incidence of acute pancreatitis can be attributed to many factors and hence it is usually caused by multifactorial risk factors which combine from the initial damage to full blown symptoms of acute pancreatitis in a span of few years. The various causes attributed to the onset of acute pancreatitis has been studied extensively and has led to an intensive research in prevention of progression of acute pancreatitis to full blown chronic pancreatitis leading to increased mortality and debilitating illness caused due to complete destruction of the pancreas anatomy. Acute pancreatitis has become a treatable entity nowadays due to the advancement of research and study of various treatment modalities which has been found effective in curing the symptoms and halting the progression of the destruction of pancreas parenchyma. The treatment modalities which have been found effective in acute pancreatitis do not offer a permanent solution for the treatment of this condition

ACKNOWLEDGEMENT

I owe my reverential gratitude and humble thanks to Lord God Almighty for all his mercy, for being with me and showering abundant blessing upon me throughout the course of the study.

I am obliged to record my immense gratitude to **Dr.B.ASHOKANMch,** The Dean , Coimbatore Medical College Hospital for providing all the facilities to conduct the studies.

I express my deep sense of gratitude and heartfelt thanks to Professor **Dr. V. ELANGO, M.S,** Head of Department of General Surgery for his dynamic guidance , constant help and encouragement throughout the study.

I express my respectful gratitude and indebtedness to my guide Professor **Dr. D.N. RENGANATHAN, M.S,** for his valuable guidance and support.

I would like to express my sincere thanks to Professor **Dr.NatarajanM.S, Professor Dr. Balasubramaniam, Professor Dr.Shanthi, Professor Dr. Nirmala**. I deeply thank **Dr. V.S. VenkadesanM.S, D.A, Dr. Jayakumar M.S., Dr.RadhikaM.S, Dr.A.Balamurugan M.S.**assistant

professors of surgery, for all the needful help they have provided for the study.

I acknowledge my gratitude to our Registrar **Dr.NarayanamoorthyM.S** and all my assistant professors of Department of surgery for their encouragement and support.

I am thankful to The ETHICAL COMMITTEE of Coimbatore Medical College for permitting me to proceed with this dissertation.

Lastly I am grateful to all the patients whose cooperation made this work possible.

DATE: Signature of the Candidate

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LIST OF ABBREVIATIONS USED

1.	BISAP	-	Beside index of severity in acute pancreatitis
2.	CTSI	-	CT severity index
3.	SAP	-	Severe acute pancreatitis
4.	PAN NEC	-	Pancreatic necrosis
5.	CEA	-	carcinoembryonic antigen
6.	SD	-	Stadard deviation
7.	CECT	-	Contrast enhanced CT Scan

INTRODUCTION

Acute pancreatitis has become a common disease among the new world population due to the increase in the socio-economic factors affecting the general population which has led to an increase in risk factors associated with the onset of acute pancreatitis. Acute pancreatitis has become a formidable cause of mortality and morbidity in recent times due to its sheer clinical features and debilitating symptoms. The increased incidence of acute pancreatitis can be attributed to many factors and hence it is usually caused by multifactorial risk factors which combine from the initial damage to full blown symptoms of acute pancreatitis in a span of few years.

The various causes attributed to the onset of acute pancreatitis has been studied extensively and has led to an intensive research in prevention of progression of acute pancreatitis to full blown chronic pancreatitis leading to increased mortality and debilitating illness caused due to complete destruction of the pancreas anatomy.

Acute pancreatitis has become a treatable entity nowadays due to the advancement of research and study of various treatment modalities which has been found effective in curing the symptoms and halting the progression of the destruction of pancreas parenchyma. The treatment modalities which have been found effective in acute pancreatitis do not

offer a permanent solution for the treatment of this condition but they prevent progression of the destruction of the pancreas and allows the body to rejuvenate from the systemic illness caused by the inflammation of the pancreas.

The only effective treatment modality which has been discovered till date is the removal of the risk factors which initiated the event of acute inflammation in the pancreas. Most commonly the incidence of gall stones has increased which has led to an increased incidence of acute pancreatitis and hence gall stones have been named as the most common cause of acute pancreatitis. Hence the treatment has been very simplified in terms of Endoscopic Retrograde Cholangio-Pancreaticography (ERCP) and retrieval of the CBD stones which leads to the removal of obstruction of pancreatic duct thereby relieving the inflammation of the pancreas.

The risk factors of acute pancreatitis should be eliminated from the initial stages of the disease to prevent disease progression and it has been proven to be the most effective treatment modality for acute pancreatitis. Evaluation of risk factors of pancreatitis has become a pivotal point in the research of the field of pancreatic pathology and hence this study has been undergone to evaluate various risk factors of acute pancreatitis and their role in the progression of this disease.

AIMS AND OBJECTIVES OF THE STUDY

1. **TO EVALUATE THE CAUSES OF ACUTE PANCREATITIS AND TO IDENTIFY THE COMMON CAUSES LEADING TO ACUTE PANCREATITIS IN OUR STUDY POPULATION.**

2. **TO IDENTIFY THE SEVERITY OF ACUTE PANCREATITIS AND TO CATEGORIZE THE TREATMENT GIVEN IN OUR STUDY POPULATION.**

REVIEW OF LITERATURE

HISTORICAL REVIEW:

- In Greek Pan means “all” and Kreas means “Flesh” and it was described first by Herophilus Chalcedon.
- Rufees and Ephesees – named the pancreas.
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ANATOMY OF THE PANCREAS

Pancreas is a retroperitoneal organ and it lies behind the stomach , transverse colon and the mesocolon. The whole organ measures over 15 cm long weighs about 90 to 120g in adult, soft in consistency with lobulated surface. It occupies the supracolic and partly the infracolic compartment. It comprises of the head , neck , body and tail.

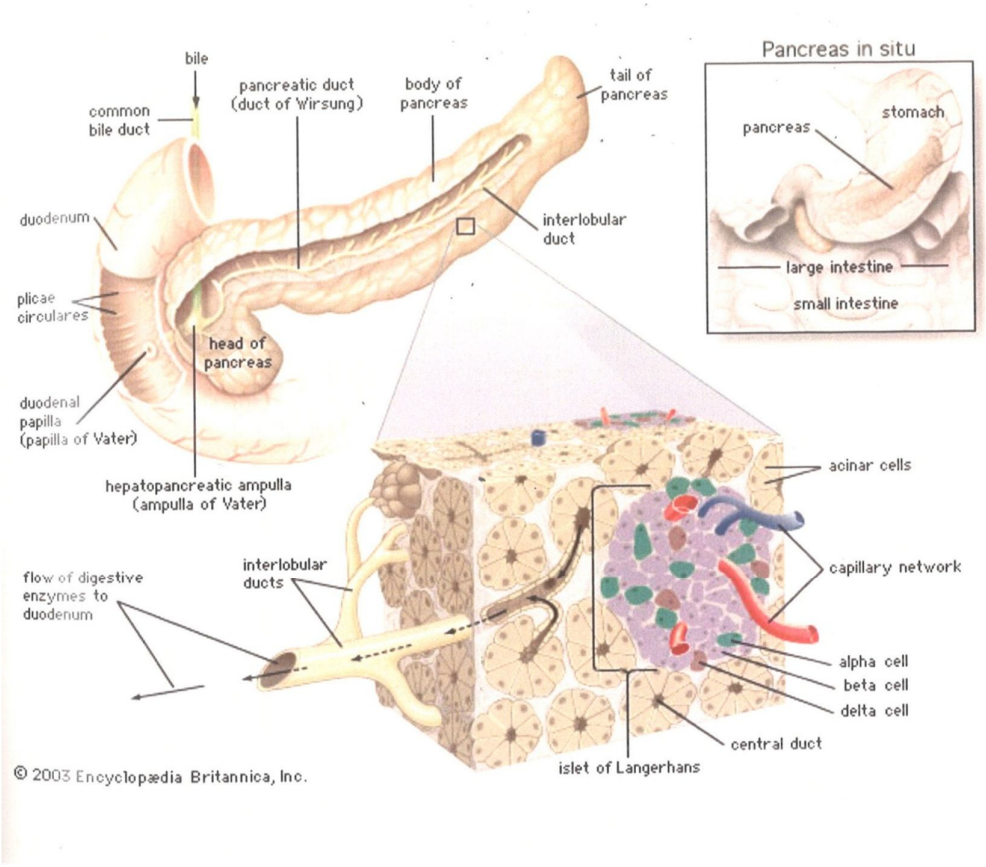
Head :

It is the broadest part , occupies the concavity of the duodenum , and lies over the inferior venacava , right and left renal veins. Its posterior surface is indented by last part of the common bile duct. The uncinate process is the wedge shaped lower part of the gland , lies posterior to the superior mesenteric artery and vein and lies anterior to the aorta , at the level of L2.

Neck :

It is the continuation of the upper part of the head , lies anterior to the superior mesenteric vein and portal vein formation. It lies at the level of L1 vertebra.

FIG 1 ANATOMY OF PANCREAS



Body :

The body starts from the neck and it runs across the left renal vein , left crus of the diaphragm, aorta, left psoas muscle, hilum or the left kidney and the lower pole of the left supra renal gland. The Splenic artery passes along the upper body of the tail and it is tortuous in its course. Splenic vein gets its tributary from the inferior mesenteric vein behind the body of the pancreas. The transverse mesocolon is attached in the lower part of the anterior surface of the body and neck.

Tail :

It passes forward from the anterior surface of the left kidney at the level of the hilum , accompanied by the splenic artery , splenic vein and lymphatics in the two layer of lieno renal ligament and then touches the hilum of the spleen.

Ductal system of the pancreas :

The duct of Wirsung is the major duct comes from the tail to the head , arises from the confluence of numerous small ducts of the lobules crossing the gland forming a “Herring bone” pattern , gradually increasing in diameter upto 10mm joins with the common bile duct in a dilatation , the ampulla of Vater , which opens into the duodenal papilla. The accessory pancreatic duct drains the uncinated process and lower part of the head of pancreas lies more on ventral plane , opens into the duodenum 2 cm proximal to the major papilla and 7 cm distal to the pylorus. Injury to the duct of Santorini in the pancreatic divisum during gastrectomy results in severe hemorrhagic or recurrent pancreatitis.

Blood Supply :

Blood supply is chiefly derived from the splenic artery which supplies the neck , body and tail by a large branch named as “ arteria

pancreatica magna “. The head is supplied by superior pancreatico duodenal artery (a branch of superior mesenteric artery) . The right hepatic artery is a branch of superior mesenteric artery , passes behind the head of the pancreas or within the substance.

Venous drainage is by small veins into the splenic vein and the head of the pancreas drains into the superior pancreatico duodenal vein into the superior mesenteric vein which forms a landmark during pancreatic dissection.

Lymphatic drainage :

Lymphatic drainage generally follows venous drainage in all directions.

They drain into the following group of lymph nodes :

A. Superior nodes drain the anterior and superior half of the gland.

B. Inferior nodes drain the anterior and posterior lower half.

C. Anterior nodes drain the anterior surface of the head of the pancreas.

D. Posterior nodes drain the posterior surface of the head.

E. Splenic nodes drain the tail of the pancreas.

Every group of lymph nodes finally drains into the coeliac and superior mesenteric group of lymph nodes.

Nerve supply :

The afferent pain sensation from the pancreas is conducted through the sympathetic fibres from the greater , lesser and lowest splanchnic nerves via the central ganglia. The celiac branch of the right vagus nerve provides the para sympathetic supply.

Development of the pancreas :

Pancreas develops as two separate buds each an outgrowth of the endoderm at the junction of foregut and midgut. The ventral bud grows into the ventral mesogastrium in common with the outgrowth of bile duct and the dorsal bud grows into the dorsal mesogastrium. The duodenal portion of the duct rotates and becomes adherent to the posterior abdominal wall , the ventral bud rotates and fuses with the dorsal bud at 7 to 8 weeks of gestation. The dorsal pancreatic duct by connecting with the ventral pancreatic duct becomes the major duct of Wirsung draining the body and tail , the proximal end is retained as accessory pancreatic duct of santorini. This duct opens into the duodenum separately in 70% of the cases and in 5% of cases it becomes the major duct – pancreatic divisum.

The pancreatic alveoli developed by the growth of cells from the terminal part of the branching ducts. The islet cells appear to have as identical origin but become separated from their parent ducts and undergo change of secretory function.

Microscopic anatomy :

This lobulated gland composed of alveoli of serous cells with , very few ducts without islet cells by characteristic staining reaction. In each alveolus the basal part of the cell is deeply stained and basophilic , while the central part is acidophilic. The nucleus is situated towards the basal part. The ducts are lined with simple columnar epithelium.

The islets , in section appear as pale areas , more prevalent in the tail with Zankes – formal fixation. It varies in size from one to four times that of pancreatic alveolus. Alpha cells produce glucagon which is situated more in the periphery of the islets , constitute 18 to 25% of the cells. Beta cells producing insulin has secretory granules , density of which varies in patient to patient. The Delta cells producing somatostatin constitutes 3 to 8 % located near the alpha cells contains granules demonstrated by electron microscopy.

FIG 2 DEVELOPMENT OF PANCREAS

PHYSIOLOGY

The pancreas has both the endocrine and exocrine functions. Exocrine pancreas– the acinar cells of the pancreas secrete enzymes and small amount of electrolyes. The centro acinar and ductular cells secrete water and electrolytes.

Composition – Total volume – 1500 to 2000 ml/day

Protein – 5 to 8 gmpH – alkaline (8.3)

It is iso-osmotic and alkalinity is due to the bicarbonate concentration which depends on the secretory rate (100 to 150 mmol/L). The Na^+ and K^+ concentration is similar to the plasma but other anions and chlorides are inversely related to the bicarbonate concentration are flow dependent.

Proteins in pancreatic juice :

1. Amylolytic enzymes – alpha amylase
2. Proteolytic enzymes
    a. Endopeptidases – Chymotripsinogen , Trypsinogen , Proelastases
    b. Exopeptidases – Procarboxypeptidase

3. Lipolytic enzymes – lipase

4. Other enzymes – Phospholipase A , carbonyl ester hydrolase , Ribonuclease Deoxyribonuclease

5. Other proteins – Immuno globulins , Lactoferrin , CEA

REGULATION OF SECRETION

Both nervous and hormonal control.

I . Cephalic Phase

Stimuli similar to gastric secretion.

Efferent fibres – Vagus nerve

Volume of secretion – small

Enzyme – High

Hormone – Gastrin from antrum.

II. Gastric Phase

Secretion further stimulated.

Both nervous and humoral control.

Distension of body of stomach excite tension in the wall of the Vasovagal reflex and causes increased enzyme output , Gastrin release due to chemical or mechanical stimuli which produce enzyme rich small volume secretion.

III. Intestinal phase

Acid chime enters the duodenum and causes the release of hormone secretin from the endocrine cells of the mucosa. Secretin stimulates watery secretion and an iso osmotic solution of bicarbonates.

Pancreozymin hormone from the I cells in crypts and villi of duodenum and jejunum on release stimulates enzyme rich secretions.

ACUTE PANCREATITIS

Defined as pancreatic inflammation followed by clinical and biological restitution of gland if the primary cause is eliminated. Different stages are distinguished in the development of acute pancreatitis. There are a number of known and unknown etiological factors capable of initiating pancreatic inflammation in a variety of ways that finally results in pancreatic necrosis.

Pancreatic involvement may be confined to the initial damage and may cease spontaneously or give rise to an activation of digestive enzymes within the pancreas thereby self perpetuating pancreatic auto digestion with fat necrosis and hemorrhage.

ETIOLOGICAL FACTORS

A number of factors either acting alone or a combination of them may be responsible for the pancreatic onslaught, they can be :

I. METABOLIC :

a. Alcohol

b. Hyperlipoprotenemia

c. Hypercalcemia

d. Drugs

e. Scorpion venom

II. MECHANICAL :

a. Cholelithiasis

b. Post operative (gastric , biliary)

c. Post traumatic

d. Obstruction of the duct

e. Pancreatic tumor

f. Duodenal obstruction

III. VASCULAR :

a. Post operative (cardio pulmonary bypass)

b. Polyarteritis nodosa

c. Atheroembolism

IV. INFECTIONS :

a. Mumps

b. Coxsackie virus infection.

DEVELOPMENT OF ACUTE PANCREATITIS :

Etiological factors described above initiate the process of bile reflux and causes the pancreatic injury. The pancreatic injury is manifested as edema , vascular injury , and pancreatic acinar damage. This injury causes the activation of the pancreatic enzymes such as trypsin , phospholipase A etc. This leads to autodigestiion and pancreatic necrosis.

MECHANISM BY WHICH COMMON ETIOLOGICAL FACTORS CAUSE ACUTE PANCREATITIS :

A) ALCOHOL – MECHANISM OF INJURY :

a. Pancreatic exocrine hypersecretion in the presence of partial ampullary obstruction.

b. Alcohol is a stimulant of gastric acid secretion and the resultant duodenal acidification releases secretin which increases the exocrine pancreatic secretion of water and bicarbonate.

c. Alcohol also increases the resistance of sphincter of Oddi causing partial obstruction to the flow of pancreatic secretion.

d. Alcohol increases the intraductal pressure in pancreatic ducts and also increases permeability of ducts to macro molecules.

e. Alcohol initiates enzyme extravasation and cause pancreatic injury as a result of protein obstruction of the pancreatic duct.

f. Intermediate state of hyper triglyceredemia following alcohol ingestion. Toxic levels of free fatty acids , produced from the lipolysis of triglycerides may cause acinar cell or capillary endothelial cell injury in the pancreas.

B) GALL STONES

Mechanism :

Gall stone migration through the ampulla of Vater , causes diversion of bile into the pancreatic duct and subsequent bile induced pancreatic parenchymal injury.

The evidences are:

Presence of gall stones in stools of 90% of patients with acute gall stone pancreatitis.

Cholangiographic studies show a common channel between CBD and pancreatic duct in 90% of patients with gall stone pancreatitis.

Intraoperative cholangiogram after cholecystectomy shows that pancreatic duct reflux in 60% of the patients with history of pancreatitis.

Endoscopic recovery of stones impacted at the Ampulla of Vater within 48 hours of onset of symptoms.

C) HYPERLIPOPROTEINEMIA

Pancreatitis associated with various primary hyperlipoprotenemic conditions are as follows :

1. Fredrickson – Type I – 30 % of occurrence of pancreatitis.
2. Fredrickson – Type IV – 15% of possibility of pancreatitis.
3. Fredrickson – Type V – 27 – 41 % of possibility of pancreatitis.

Since type IV is the commonest form of Hyperlipoprotenemia , this accounts for most examples of lipid associated pancreatitis. Free fatty acids released by pancreatic lipase may exert a toxic influence as the pancreatic parenchyma.

D) HYPERPARATHYROIDISM AND HYPERCALCEMIA

Incidence – 7-19%

Mechanism :

Calcium induced trypsinogen activation and subsequent auto destruction.

Calcium associated stone precipitation in the duct causing obstruction.

Calcium stimulated pancreatic exocrine hypersecretion.

Direct toxic effect on parenchyma of pancreas by parathormone.

MECHANISM OF ACUTE PANCREATITIS

1. INTRA PANCREATIC ACTIVATION OF PANCREATIC ZYMOGENS

The cardinal mechanism is the activation of the trypsinogen to trypsin and this enzyme activates the other enzymes and the pathology continues. Whatever may be the etiology finally it lands upon the above given mechanism.

A concept known as the intrapancreatic activation of the enzymes is postulated. The release of the pancreatic enzyme is hindered and they join the intracellular lysosomes and this results in activating all known pancreatic zymogens like chymotripsinogen to active chymotrypsin , proelastase to elastase and prophospholipase to lipase A. Only lipase already synthesized in active form is independent of trypsin. Every activated enzyme has its own function and it is summarized in the flow chart.

Of all the etiological factors alcohol is the most common cause of acute pancreatitis so its mechanism is discussed. The mechanism is as follows :

Hyper secretion of the exocrine pancreatic secretion in the presence of partial ampullary obstruction.

Enzyme extravasations initiating the pancreatic injury.

Alcoholics usually have hypertriglyceridemia this also initiates pancreatitis.

The next common cause is the gallstone pancreatitis. Gall stone migrates into the ampulla of Vater which causes the diversion of the bile into the pancreatic duct which results in the bile induced pancreatic injury.

PATHOLOGICAL CHANGES IN ACUTE PANREATITIS :

Mildest pathological change - Edema of the gland. May be accompanied by infiltration of the intra lobular septa by inflammatory cells.

Microscopy - fat necrosis in the pancreas and surrounding tissues.

If extensive necrosis - Whitish yellow plaques occur due to necrosis and calcium deposition.

Vascular thrombosis or disruption results in pancreatic necrosis or gross hemorrhagic infarction.

Increased levels of active pancreatic enzymes occur :

1. Within the pancreas

2. In the peritoneal exudate

3. In the blood stream of the patients with acute pancreatitis.

FIG 3 PATHOPHYSIOLOGY OF PANCREATITIS

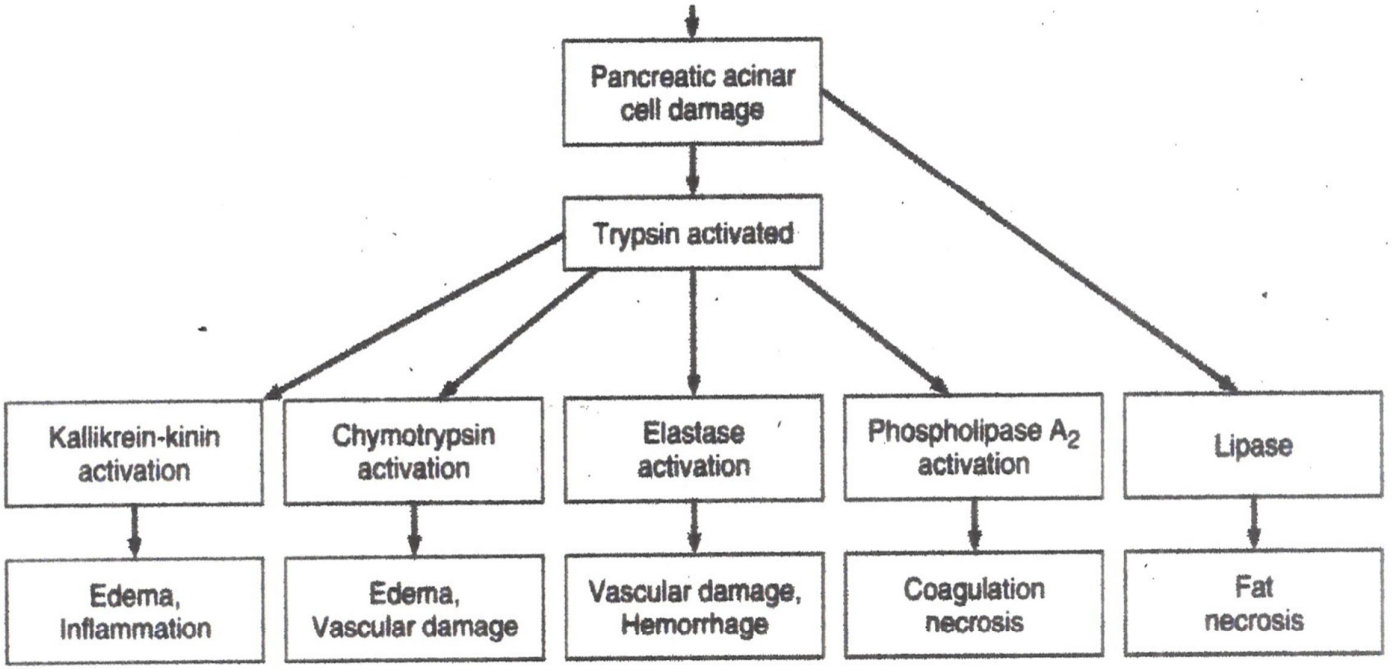
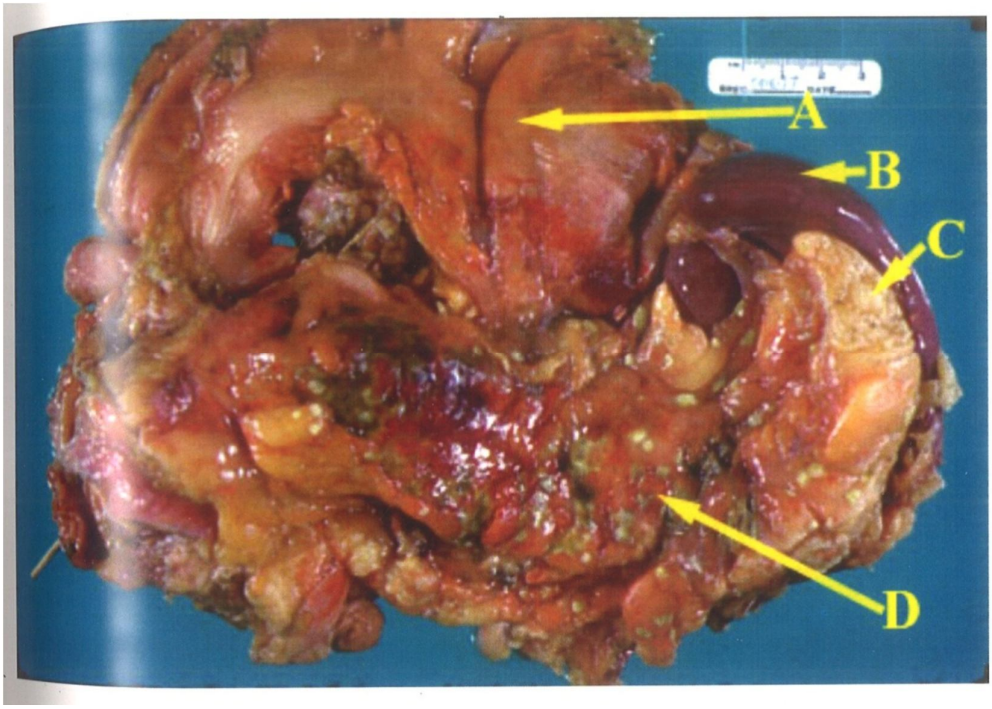


Figure 15–3. Hypothesized pathogenesis of acute pancreatitis. (Reproduced with permission from Marshall JB: Acute pancreatitis: A review with an emphasis on new developments. Arch Intern Med 1993;153:1185.)

FIG 4 ACUTE PANCREATITIS SPECIMEN



The specimen shows acute pancreatitis with severe necrosis

CHANGES IN VARIOUS SYSTEMS OF THE BODY IN ACUTE PANCREATITIS:

The pathophysiology alters many systems in the body. The changes affects the following systems:

1. Fluid and electrolyte changes.

2. Cardiovascular changes.

3. Respiratory changes.

4. Renal changes.

5. Local changes.

FLUID AND ELECTROLYTE CHANGES :

Circulating blood volume is decreased due to loss from intravascular space of plasma into the retroperitoneum and systematically. Additional loss occurs following vomiting or naso gastric aspiration. Hypocalcemia and Hypomagnesemia are frequent. Decreased ionised calcium levels also occur due to trapping of calcium in areas of fat necrosis.

CARDIOVASCULAR CHANGES :

Hypotension , Tachycardia , increased total peripheral vascular resistance and decreased cardiac output - sequelae of hypovolemia also observed in acute pancreatitis similar to septic shock or hepatic cirrhosis are due to circulatory vasoactive substances. Hypotension persists despite restoration of intravascular volume.

RESPIRATORY COMPLICATIONS :

- Early features of acute pancreatitis - arterial hypoxemia
- Pulmonary function studies - Decreased respiratory lung volume with decreased pulmonary compliance and decreased diffusion capacity.
- Early respiratory failure resolves with subsidence of acute pancreatitis.
- Severe or unresolving pancreatitis may develop progressive pulmonary insufficiency , infiltrates and pleural effusion.

FACTORS IMPLICATED FOR PULMONARY COMPLICATIONS:

1. Abdominal distension and elevation of diaphragm.
2. Alteration in the lecithin of pulmonary surfactant by circulating pancreatic lecithinase.
3. Pulmonary thromboembolism.
4. Circulating free fatty acids.
5. Circulating products of the proteolyitc cleavage complement.

RENAL FAILURE :

Major factor for the cause of death in patients with acute pancreatitis. Due to principally hypovolemia. Therefore , many patients land up in acute renal failure. Pathologically it is due to deposition of the fibrin complexes in the glomeruli.

OTHER SYSTEMIC FEATURES :

Abnormal liver function tests - elevation of serum bilirubin and liver enzymes such as Alkaline phosphatase are raised and it is mainly due to biliary obstruction and pericholangitis.

Early intravascular thrombosis with decreased platelet count and fibrinogen level occur due to the effects of pancreatic proteolytic enzymes. May be followed by marked thrombocytosis and hyperfibrinogenemia.

LOCAL SEQUELAE :

Intra abdominal complications include :

1. Paralytic ileus

2. Duodenal / biliary obstruction

3. Release of pancreatic enzymes with peripancreatic fluid collection and fluid in general peritoneal cavity.

4. Destruction of tissues adjacent to pancreas.

5. Rarely cause gross disruption of the pancreatic ductal system which is usually self limited.

6. Persistent chronic pseudocyst in 1% of the patients.

7. Infected pancreatic abscess due to secondary infection occur in 1-9% patients and organisms are usually enteric.

8. Extension of local necrosis to involve colonic wall causing colonic perforation occurs in 1% of the patients and occurs in the left transverse colon or splenic flexure.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

The classical feature of acute pancreatitis is its severity of symptoms and paucity of physical signs.

1. Abdominal pain - 85 - 100%

Upper abdominal pain may radiate to the back and may be severe. Pain is aggravated by intake of food or intake of alcohol. Pain is resistant to analgesics. Patient assumes various postures in an effort to obtain relief from the pain.

2. Nausea and vomiting - 92%

Vomiting is usually non projectile and it of low volume and it contains gastric and duodenal content and it is non feculent.

3. Physical examination :

- Restless patient.
- Rapid pulse and respiratory rate

- Arterial hypotension
- Abdomen - moderately distended with epigastric dullness. Tenderness is marked in the upper abdomen.
- Moderate muscle spasm is present.
- **GREY TURNERS SIGN -** Grey green discoloration of the flank present in patients with peripancreatic hemorrhage.
- **CULLEN'S SIGN -** bluish discoloration of periumbilical region.

4. Extra abdominal manifestations :

- Left sided pleural effusion
- Acute pulmonary failure marked by Tachypnoea and dyspnoea.
- Central or peripheral cyanosis due to :

  a. Circulating Phospholipase A

  b. Circulating free fatty acids from triglycerides from lipolysis

  c. Pulmonary surfactant loss

  d. Volume overload with pulmonary capillary leakage.

5. Central Nervous System manifestations -

Nonlateralizing nature , including billigerence , confusion , psychosis and coma. This is due to hyperosmolarity , hypoperfusion , hypoxia , cerebral fat embolism or disseminated intravascular coagulation.

LABORATORY INVESTIGATIONS :

DIAGNOSIS OF ACUTE PANCREATITIS :

LABORATORY TEST	RADIOGRAPHIC PROCEDURES
• Serum Amylase	• Chest X-ray
• Serum Amylase isoenzymes	• Plain Abdominal X-ray
• Urine Amylase	• Ultrasonography
• Amylase-Creatinine clearance Ratio	• Contrast-Enhanced CT Scan

1. BLOOD COUNT :

- Leucocytosis - 10,000 to 20,000 occurs early in all cases
- Hematocrit - is high in most patients at the onset
- Hemoglobin decreased value of more than 2.5gm% without detectable blood loss is found in patients with pancreatic necrosis.

2. SERUM AMYLASE :

Elevated in 95% of patients with Acute pancreatitis. But this is not an ideal marker because it is elevated in other conditions such as :

- Perforated peptic ulcer
- Biliary Lithiasis
- Intestinal obstruction
- Mesenteric Infarction.

Also in patients with acute pancreatitis , serum amylase in normal levels can occur due to :

- Hyper triglyceredemia - Latescent serum
- Assayed 3 days or more after onset.

- Previous attach has destroyed most glandular tissues.
- Present attack is associated with massive destruction of gland.

Serum amylase in Acute pancreatitis is elevated within 24 hours of onset of symptoms and returns to normal in 7 days.

3.SERUM ISOAMYLASE - P :

As it is produced only from pancreas it has a higher specificity in detection and confirmation of acute pancreatitis.

4.SERUM LIPASE :

Serum lipase is solely of pancreatic origin hence serum lipase level is more specific than amylase. Recent development of an enzyme immunoassay of lipase is reliable and is of great value in Acute pancreatitis. Duration of Hyper Lipasemia exceeds hyper amylossemia.

5. PLUERAL AND PERITONEAL FLUID AMYLASE :

Pleural effusion shows raised levels of amylolytic activity in pancreatitis. High activities of amylase may also be found in fluids aspirated from peritoneal cavity in patients with Acute pancreatitis.

6. OTHER BIOCHEMICAL INDICES :

- Hyperglycemia and Glycosuria - Non specific , transient cause - relative hypoinsulinemia and Hyperglucognemia

- Hypocalcemia - Well recognised entity in acute pancreatitis but can also occur in perforated peptic ulcer.

Cause - Deposition of calcium in areas of fat necrosis. Release of glucagon , Inadequate parathyroid response and Dilutional hypo albuminemia.

- Methemalbumin :

  Appearance in serum indicates necrotic rather than edematous pancreatitis.

- Liver function tests :

  Slight increases in alkaline phosphatase and amino transfer are with raise in serum bilirubin - transitions. Markedly elevated serum aspartate and alanine amino transferase are within 48 hours after onset discriminates biliary from non biliary pancreatitis.

RADIOGRAPHIC FINDINGS :

I. PAIN X-RAYS :

i) Plain X-ray Abdomen :

a) Segmental small bowel ileus or a "SENTINEL LOOP" in the left upper quadrant.

b) Dilatation of the transverse colon - "COLON CUT OFF SIGN"

A. Increase epigastric soft tissue density.

B. Obscured psoas muscle margins.

C. Presence of gall stones.

D. Pancreatic calcification - may not be an acute pancreatitis.

ii) Plain X-Ray Chest :

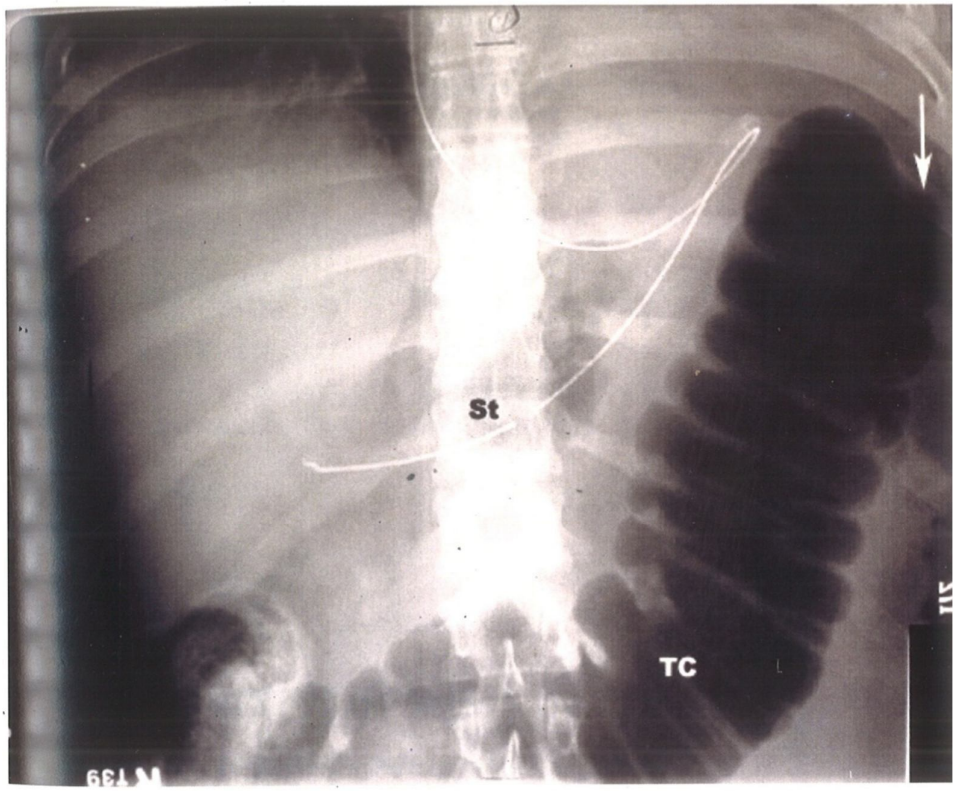
a) Pleural effusion

b) Atelectasis

c) Pneumonia

d) Pulmonary Edema.

FIG 5 X RAY SHOWING COLON CUT OFF SIGN



CONTRAST STUDIES WITH WATER SOLUBLE CONTRAST :

Upper Gastrointestinal study :

a) Widening of "C" loop

b) Anterior displacement of stomach

c) Subtle duodenal mucosal sign

ABDOMINAL ULTRASONOGRAM :

a) Enlargement and edema of pancreas

b) Pseudocysts of pancreas

c) Delineates pancreatic abscess

d) Dilatation of Bile duct and presence of stone in gall bladder and common bile duct.

COMPUTED TOMOGRAPHY :

Except in early or mild eases it is useful in assessing CT scan reveals many findings in patients with Acute pancreatitis. Pancreas is usually enlarged and there is pancreatic edema. Pancreatic necrosis is characterized CT scan. In the peri pancreatic area there is collection , obliteration of the fat plane and thickening of the fat plane. Other findings are paralyitc ileus and Pleural effusion.

ENDOSCOPY :

To detect biliary pancreatitis.

Therapeutically used for papillotomy and removal of stones impacted at the ampulla of Vater.

FIG 6 ACUTE PANCREATITIS WITH PANCREATIC EDEMA

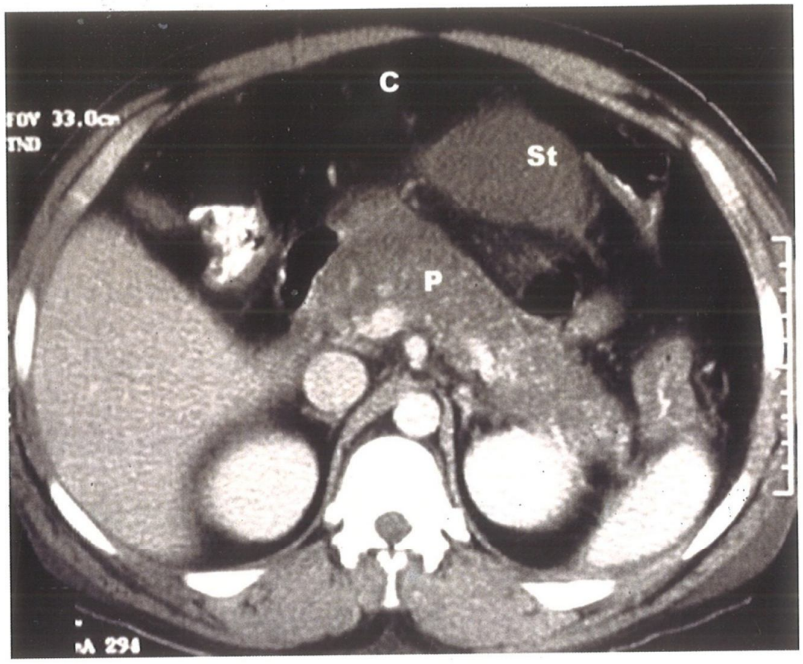


FIG 7 SEVERE NECROTIC PANCREATITIS

TREATMENT

MEDICAL MANAGEMENT :

A. NUTRITION :

1. Enteral nutrition :

Previously it was thought that enteral nutrition stimulates the pancreas and results in pain in pancreatitis , but now it is found that pancreas is actually in a state of rest in acute pancreatitis so it better to stimulate the pancreas. So enteral feeding does no harm to the patient.

2. Total parenteral nutrition :

TPN is associated with many complications such as arterial injury , pneumothorax , thrombosis , and catheter embolism. Many studies confirm that enteral nutrition is better than TPN.

B. GASTRITIS PREVENTION :

Patients suffering from severe AP have risk to develop peptic ulcers or erosive gastritis Histamine 2 antagonists is indicated in patients on mechanical ventilation and patients with adult respiratory distress syndrome (ARDS).

C. FLUID MANAGEMENT :

Adequate fluid management is the main stay in the management of acute pancreatitis. If missed it can lead to serious complications. Plenty of fluids are sequestered in third spaces. So crystalloids and colloids are used in the ratio of 3:1 . The fluid loss may be 6-10 litres. It is said that when hematocrit is less than 30% if dextran is used it improves the microcirculation. Good fluid resuscitation is indicated by adequate urine output , CVP around 8-12 cm of water , hematocrit of 35-40 %.

D. PAIN MANAGEMENT :

As a result of activation of pancreatic enzymes there are increase in release of the inflammatory mediators. These mediators irritate the sensory fibers of the celiac plexus (T5 TO T9) and cause severe pain radiating to the back. The following drugs are used in management of pain :

- Non steroidal analgesia
- Meperidine
- Tramadol
- Thoracic epidural analgesia.

E. THE ROLE OF ANTIBIOTICS :

There is a great controversy regarding used of antibiotics in acute pancreatitis. Since there is great risk in developing necrosis in acute pancreatitis , there is also a problem in developing abscess formation , antibiotics are used. The most common antibiotics used are imipenum , meropenum , metronidazole , fluoroquinolones and cephalosporins. These antibiotics penetrate the pancreas well , but aminoglycosides do not penetrate the pancreas. Over use of antibiotics result in fungal infection.

F. SUPPRESSION OF PANCREATIC EXOCRINE SECRETION :

These are done by nasogastric suction , histamine H-2 receptor antagnoists , antacids , atropine , glucagon , calcitonin and stomatostatin.

SURGICAL MANAGEMENT : INDICATIONS AND TIMING OF INTERVENTION :

1. Uncertainty of diagnosis
2. Treatment of pancreatic sepsis
3. Correction of associated biliary tract disease
4. Progressive clinical deterioration despite optimal supportive care.
5. Infected necrosis.

6. Severe sterile necrosis

7. Symptomatic organized pancreatic necrosis

A) BILIARY OPERATIONS IN PATIENTS WITH CHOLELITHIASIS :

a. Cholecystostomy

b. Common duct drainage

c. Cholecystectomy

d. Early endoscopic papillotomy.

In patients with severe gall stone pancreatitis early intraabdominal surgery has been associated with higher mortality than early non operative treatment. Surgical correction of cholelithiasis to prevent recurrent pancreatitis undertaken once evidence of pancreatitis has subsided usually during the same hospital admission.

B) SURGICAL MANAGEMENT : PROCEDURES

1. RESECTION

2. PANCREATIC DEBRIDEMENT

3. MINIMALLY INVASIVE APPROACHES

- Retroperitoneal approach via dorsal lumbotomy

- Percutaneous necrosectomy and sinus tract endoscopy

RESECTION :

Pancreatic resection is primarily of historical interest only and is not recommended currently.

PANCREATIC DEBRIDEMENT :

All pancreatic debridement and post debridement care based on :

1. Wide removal of devitalized and necrotic tissue.
2. The assurance of post operative removal of the products of ongoing local inflammation and infection.

TECHNIQUE OF DEBRIDEMENT :

1. Debridement and closed drainage.
2. Open packing for pancreatic necrosis.
3. Debridement and continuous Closed post operative Lavage of the Lesser Sac.

DIFFERENTIAL DIAGNOSIS :

In addition to the diagnosis of acute pancreatitis , the general surgeon should also have a clinical suspicion of the various other disease entities that can occur along with acute pancreatitis or conditions that can

mimic acute pancreatitis and can cause a clinical dilemma in the diagnosis of Acute pancreatitis.

Various differential diagnosis of Acute pancreatitis are :

- Acute Mesenteric Ischemia
- Acute Respiratory Distress Syndrome
- Bacterial Pneumonia
- Cholangitis
- Cholecystitis
- Chronic Pancreatitis
- Colon Cancer
- Colonic Obstruction
- Community-Acquired Pneumonia (CAP)
- Emergent Treatment of Gastroenteritis
- Gallstones (Cholelithiasis)
- Gastric Cancer
- Irritable Bowel Syndrome
- Myocardial Infarction
- Pancreatic Cancer

- Pancreatic Pseudocysts
- Peptic Ulcer Disease
- Viral Hepatitis

THE COMPLICATIONS OF ACUTE PANCREATITIS :

The complications of Acute pancreatitis are :

1. Local complications :
    - Fluid collection
    - Pancreatic ascites / pleural effusion
    - Pancreatic Pseudocyst
    - Pancreatic necrosis
    - Infected pancreatic abscess
    - Hemorrhagic / Pseudo aneurysm.

2. Regional complications :
    - Venous thrombosis
    - Paralytic ileus
    - Intestinal obstruction
    - Intestinal ischemia / necrosis
    - Cholestasis

3. Systemic complications :

- Systemic inflammatory response syndrome
- Multiple organ dysfunction syndrome
- ARDS / Pulmonary failure
- Renal failure
- Cardiovascular complications
- Hypocalcemia
- Hyperglycemia
- Disseminated intravascular coagulopathy
- Protein calorie malnutrition.

A) PANCREATIC ABSCESS :

Pancreatic abscess - incidence 9%. Most common in patients with post-operative pancreatitis.

Clinical features :

- Persistent or recurrent fever
- Abdominal distension
- Abdominal mass
- Hypotension (SBP 90mmHg)

- Pneumonia / Effusion
- Renal failure
- Coma
- Elevated serum Amylase
- Leucocytosis (more than 11,000 / mm^3)

Radiographic diagnosis :

1. Upper GI contrast studies showing displacement of stomach or duodenum. Gas outside of GIT.

2. Ultrasound abdomen can delineate pancreatic abscess

3. Computed Tomography sensitive and specific.

4. Percutaneous aspiration under CT guidance.

Treatment :

Adjuvant :

1. Vigorous supportive management.
2. Meticulous respiratory care and Nutritional support.
3. Prevention of GIT hemorrhage.

Specific :

1. Percutaneous drainage by catheter.
2. Laprotomy - Debridement and packing of pancreatic bed.
3. Surgical correction of other complications like involvement of colon by colostomy.
4. Feeding jejunostomy to correct nutritional imbalance.

B) PSEUDOCYSTS :

Pseudocysts following acute pancreatitis spontaneous disappearance of pseudocysts is a common occurrence in acute pancreatitis. These are carefully monitored by serial ultrasonogram or CT and operative intervention is needed only when they go in for further complications. It is dealt in detail along with treatment of pseudocysts following chronic pancreatitis.

C) PANCREATIC ASCITES :

Pancreatic ascites - more common following chronic pancreatitis. But may also follow acute pancreatitis secondary to trauma, pseudocysts and rarely pancreatic neoplasm. Treatment is by drainage procedures as dealt in pancreatic ascites following chronic pancreatitis.

PROGNOSTIC ASSESSMENT

PROGNOSTIC INDICATORS :

Because of the variability and unpredictability of acute pancreatitis, clinical scoring systems have been made to predict the severity of acute pancreatitis.

1. RANSON'S CRITERIA

2.BISAP SCORE

3. CT SEVERITY INDEX

4. OTHER PROGNOSTIC SYSTEMS :

- The Acute Physiology and Chronic Health Evaluation II (APACHE II) Score.
- C-reactive protein (CRP) assays.
- Trypsinogen-activating peptide (TAP) assays.
- ATLANDA CLASSIFICATION :

    1. ACUTE EDEMATOUS PANCREATITS - MILDER FORM. MORTALITY - 1%

2. ACUTE NECROTISING PANCREATITIS - Incidence 20% and characterized by pancreatic necrosis. MORTALITY - 15 - 20%

GRADING OF ACUTE PANCREATITIS :

This uses Ct scan primarily to grade the severity. CT scan is used to find pancreatic changes and necrosis. Either contrast or plain CT is used. This is developed by BALTHAZAR and Acute pancreatitis is graded from A to E. It gives points according to the following criteria :

1. Nature of necrosis
2. Peri pancreatic changes.

Mild pancreatitis , Interstitial pancreatitis :

Patients with pancreatitis having no collection or necrosis. They have a mild pancreatitis. Balthazar grade A-C comes under this group. CTSI is 2

Severe pancreatitis or necrotizing pancreatitis :

They occur in 20 of patients. They have uneven clinical course and high mortality rate. They are more than fluid collections. The grade is D or E and this score is usually above 3. The peri pancreatic collection is

due to fat necrosis. This group of patients with necrosis has most complication and they have to be identified. There is a separate type called extra pancreatic necrosis which has no pancreatic necrosis the CTSI is 4.

Balthazar Scoring for the Grading of Acute Pancreatitis

Grade A – normal CT

Grade B – focal or diffuse enlargement of the pancreas

Grade C – Pancreatic gland abnormalities and peri pancreatic inflammation.

Grade D – Fluid collection in a single location.

Grade E – two or more collections and/or gas bubbles in or adjacent to pancreas.

CT severity index = CT grade point + points for necrosis

First grade points are calculated. The grade A,B,C,D, E have 0,1,2,3 and 4 respectively. The points for necrosis are based on the percentage of necrosis. When the necrosis is <30% the point is 2 , 30-50% the point is 4 , >50% the point is 6.

CT grade points are added to points assigned for percentage of necrosis to determine the CT severity index. So the patients with score greater than three is said to manifest severe disease.

METHODOLOGY

MATERIALS AND METHODS

STUDY AREA :

GOVERNMENT COIMBATORE MEDICAL COLLEGE AND HOSPITAL.

STUDY POPULATION :

Patients admitted in CMCH with symptoms suggestive of Acute pancreatitis.

INCLUSION CRITERIA :

1. Clinical findings suggestive of Abdominal pain characteristic of acute pancreatitis.

2. Elevated serum amylase

3. CT Scan with findings suggestive of acute pancreatitis.

The following criteria are studied :

1. Severity of Acute pancreatitis.

2. Pancreatic Necrosis.

3. Mortality.

DEFINITION :

1. MILD OR SEVERE AP :

Depending on the organ failure the patients are classified into mild or severe Acute pancreatitis. The presence of organ failure is again dictated by the presence of following factor :

- Pulmonary failure (Pco_2 - 60 mmHg)
- Renal failure (Serum Creatinine levels more than 2 mg/dL)
- Severe Shock.

2. PANCREATIC NECROSIS :

This finding is easily found by CECT scan. This is detected by the absence of enhancement in the CECT Scan in the pancreatic parenchyma.

EXCLUSION CRITERIA :

1. Pediatric patients were excluded from the study.
2. Pregnant and postpartum patients were excluded.

STUDY PERIOD : June 2016 to July 2017

SAMPLE SIZE : 100

All patients eligible by inclusion and exclusion criteria are to be included in this study.

STUDY DESIGN :

A RETROSPECTIVE OBSERVATIONAL STUDY

RESULTS

STATISTICAL ANALYSIS :

The data are reported as the mean +/- SD or the median , depending on their distribution.

A) SEX DISTRIBUTION :

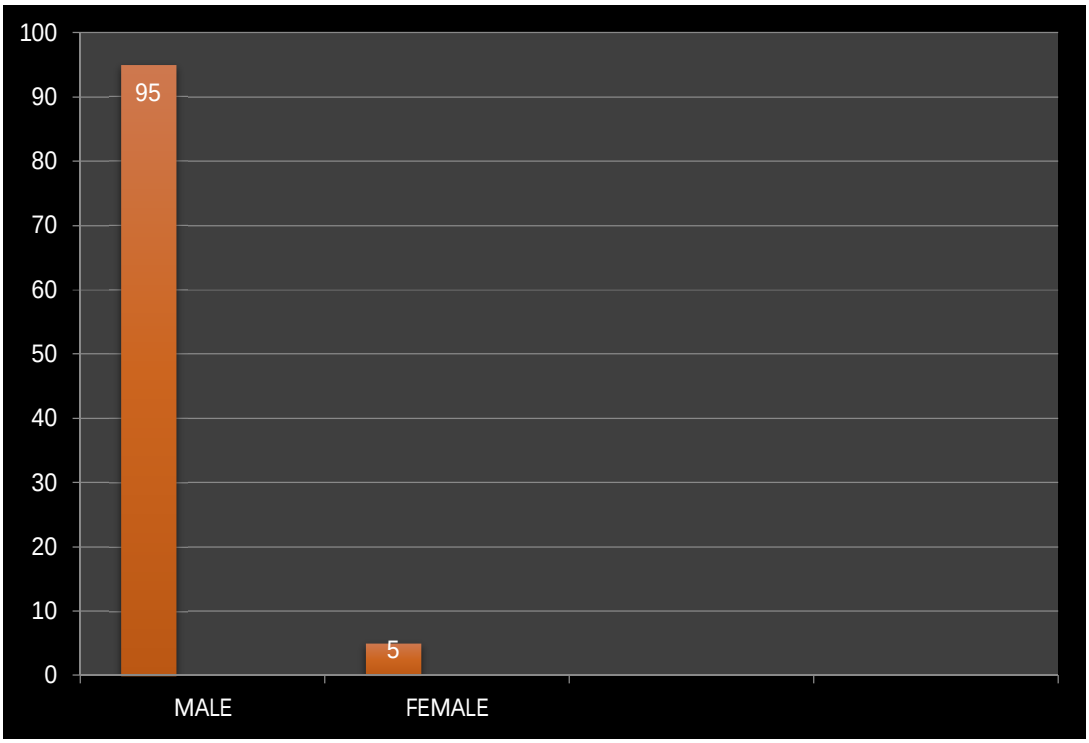
Table -1 – Sex distribution :

Male	Female
95	5

The above tabular column 1 gives the sex distribution of the disease in both the gender groups.

The total number of patients studied in the study were 100, it comprises of 95 males and 5 females.

SEX DISTRIBUTION:



The total number of patients studied in the study were 100, it comprises of 95 males and 5 females.

In our population males are commonly affected than the female population , this factor has link with the etiology. In our population alcohol consumption is the common cause of acute pancreatitis. Alcohol consumption is not prevalent in female population so female are not commonly affected by acute pancreatitis

AGE DISTRIBUTION

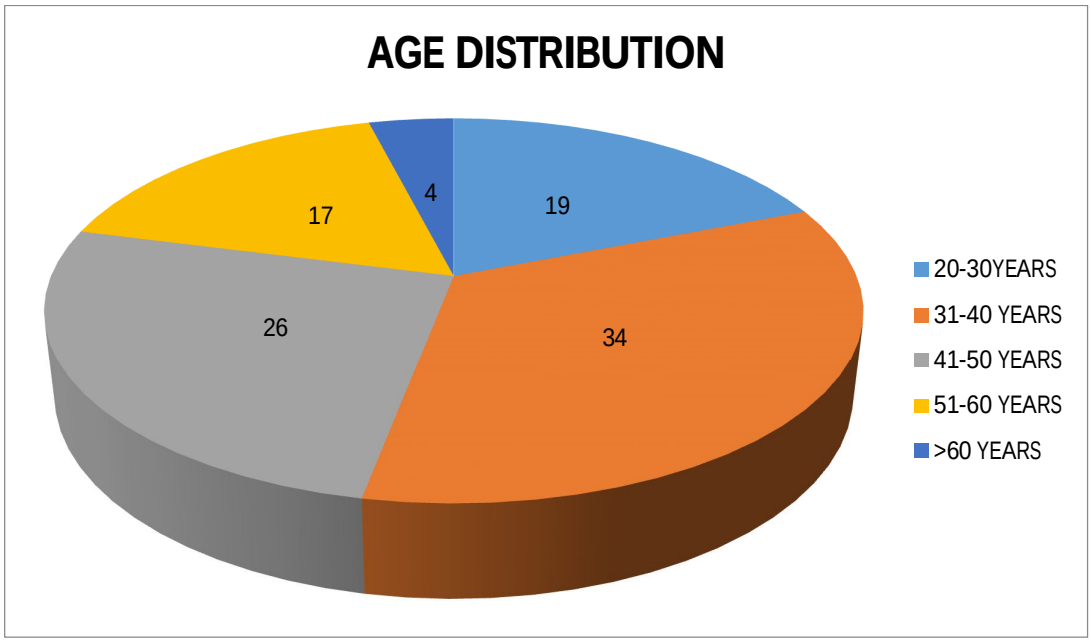
Table 2 – Age distribution

Age	Male	Female	Total
20-30	19	0	19
31-40	31	3	34
41-50	25	1	26
51-60	17	0	17
>60	3	1	4

The above tabular column 1 gives the age sex distribution of the disease in various age groups.

Maximum number of patients affected between the age group of 31-40 years and it is 34 patients.

Minimum number of patients affected in the age group of 0f > 60 years and its 4 patients



Maximum number of patients affected between the age group of 31-40 years and it is 34 patients.

Minimum number of patients affected in the age group of 0f > 60 years and its

4 patients

The pie diagram shows the same

Table -3 – Mean SD for Age

Gender	N	Mean +/- SD	Range
Male	95	39 +/- 11	20 - 66
Female	5	39 +/- 12	36 - 63
Total	100	39 +/- 11	22 - 66

The above tabular column gives the mean and the SD for the age. The explanation as follows ,

The total number of male patients in the study population are 95 with a mean of 39 +/- 11 and an age group of range 20 – 66.

The total number of female patients in the study population are 5 with a mean of 39 +/- 11 and an age group of range 36 – 63.

The overall mean of age in the study population is 39 +/- 11 and the age range of the entire study population is 22 – 66.

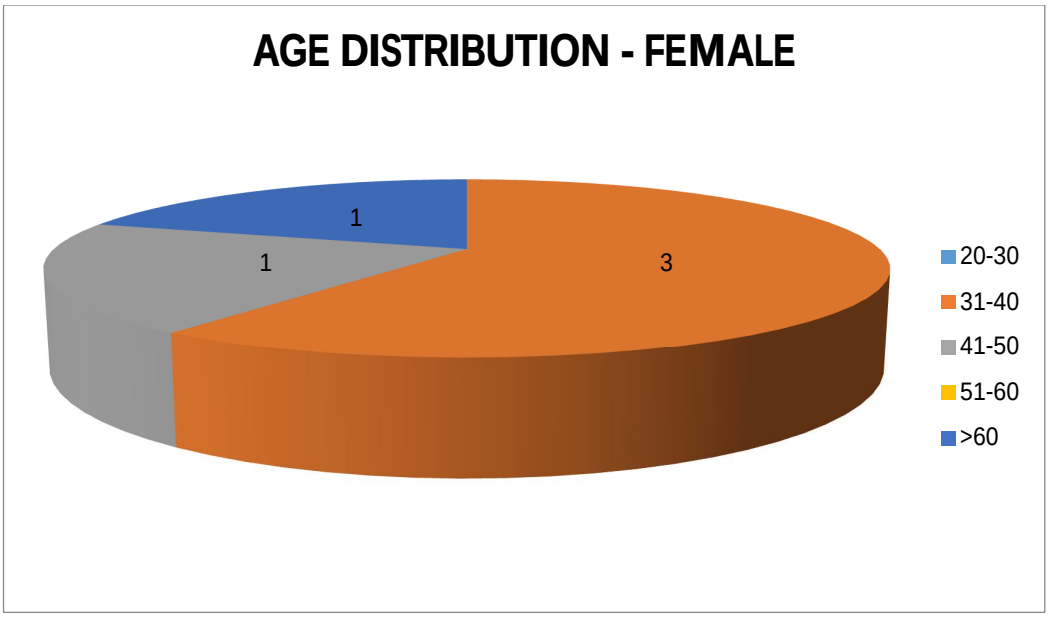
AGE DISTRIBUTION – FEMALE :

This pie diagram shows the number of patients affected with acute pancreatitis in different age group.

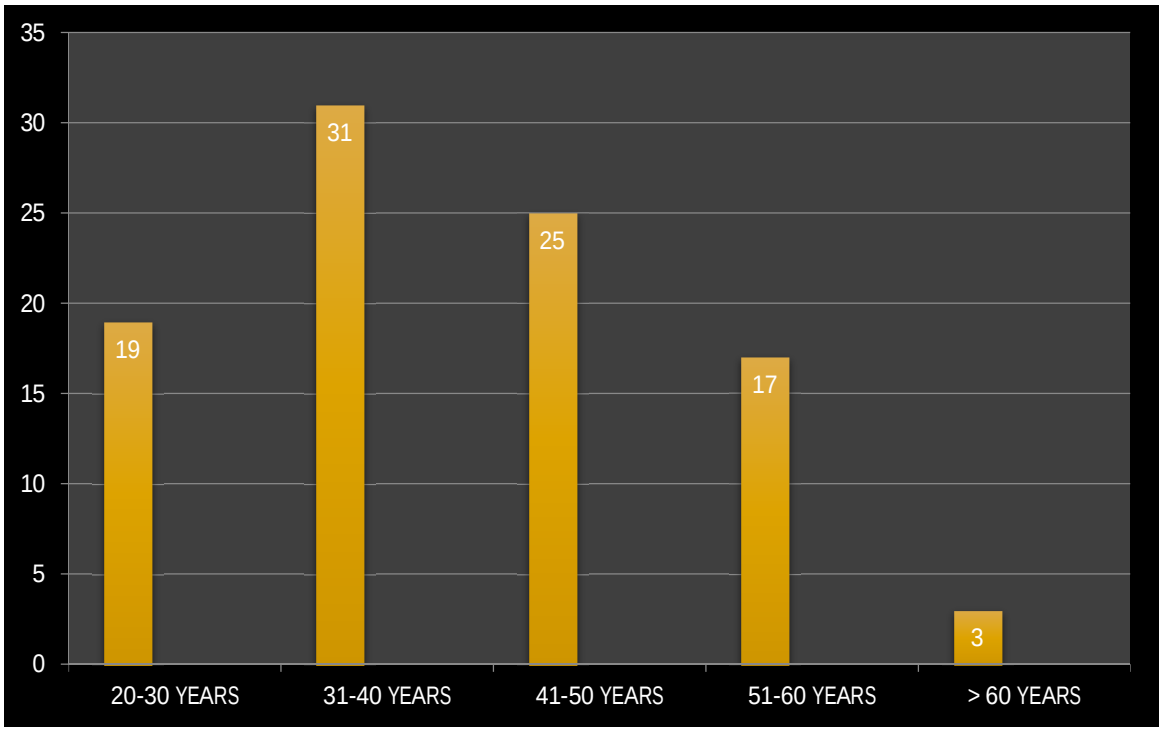
Maximum number of female patients affected between the age group of 31-40 years and it is 3 patients.

Minimum number of female patients affected in the age group of 0f 51-60 years and 21-30 years of age group of study population which is nil.

The mean age of female patients affected with acute pancreatitis in this study is 39 +/- 11.



AGE DISTRIBUTION – MALE :



This graph shows the number of patients affected with acute pancreatitis in different age group.

Maximum number of male patients affected between the age group of 31-40 years and it is 31 patients.

Minimum number of male patients affected in the age group of 0f > 60 years and its 3 patients

The bar diagram shows the same.

ETIOLOGY :

Table 4 – Etiology – Male

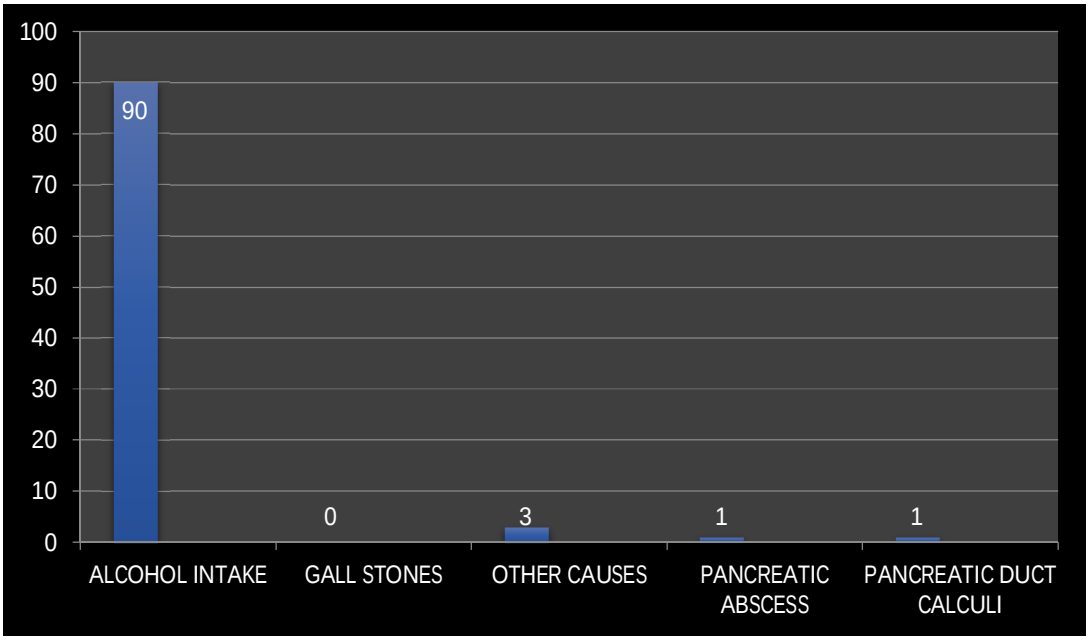
Causes	No. of cases	Percentage of study population
Gall Stones	0	0%
Alcohol	90	95%
Idiopathic	3	3%
Pancreatic abscess	1	1%
Pancreatic duct calculi	1	1%

The above tabular column gives the various etiological factors implicated in the onset of acute pancreatitis in male patients in the study population.

In our area the most prevalent cause is chronic alcohol intake.

In our study 90 male patients have history of chronic alcohol intake. 3 patients the cause of acute pancreatitis was due to idiopathic causes . The unknown causes may be due to drugs, increase in cholesterol and hypercalcemia .

CAUSES OF ACUTE PANCREATITIS - MALE :



This bar diagram shows the different causes of acute pancreatitis in male patients prevalent in our study population.

In our area the most prevalent cause is chronic alcohol intake.

In our study 90 male patients have history of chronic alcohol intake. 3 patients the cause of acute pancreatitis was due to idiopathic causes . The unknown causes may be due to drugs, increase in cholesterol and hypercalcemia . The unknown causes needs further evaluation, even in unknown causes 2 patients had history of alcohol intake during their earlier days and stopped recently for various reasons.

Table 5 – Etiology – Female

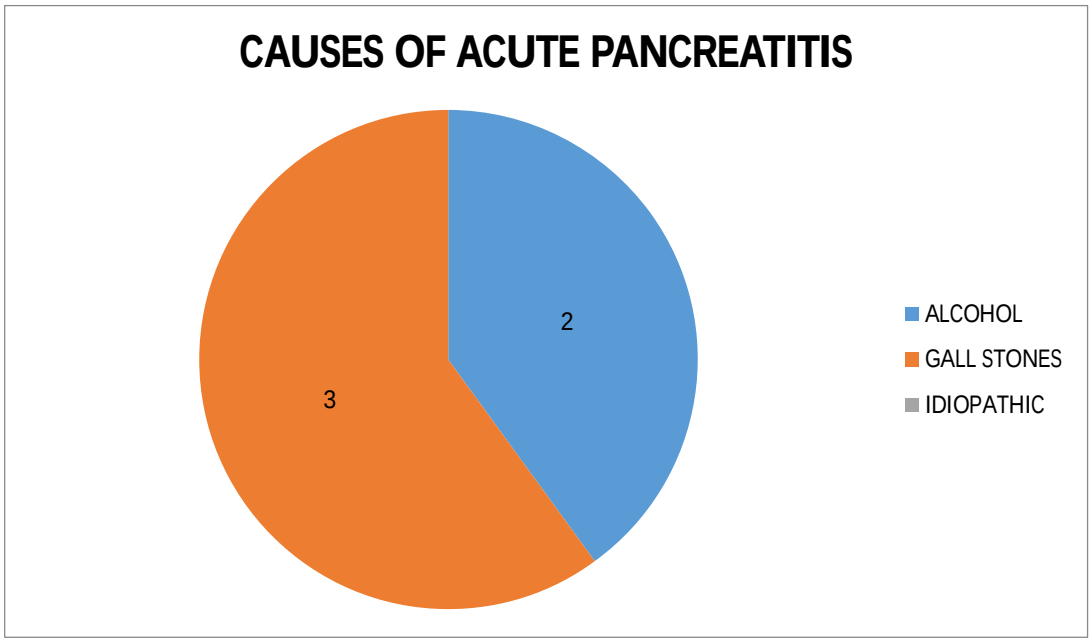
Causes	No. of cases	Percentage of study population
Gall Stones	3	60%
Alcohol	2	40%
Other causes	0	0%

The above tabular column gives the various etiological factors implicated in the onset of acute pancreatitis in female patients of the study population.

In our study 3 female patients have been diagnosed to have gall stones which caused the onset of acute pancreatitis.

In our study , 2 female patients have been diagnosed to have acute pancreatitis due to the intake of alcohol.

CAUSES OF ACUTE PANCREATITIS IN FEMALES :



This pie diagram shows the different causes of acute pancreatitis in female patients prevalent in our study population.

In our area the most prevalent cause of acute pancreatitis in female patients is gall stones.

In our study 3 female patients have been diagnosed to have gall stones which caused the onset of acute pancreatitis.

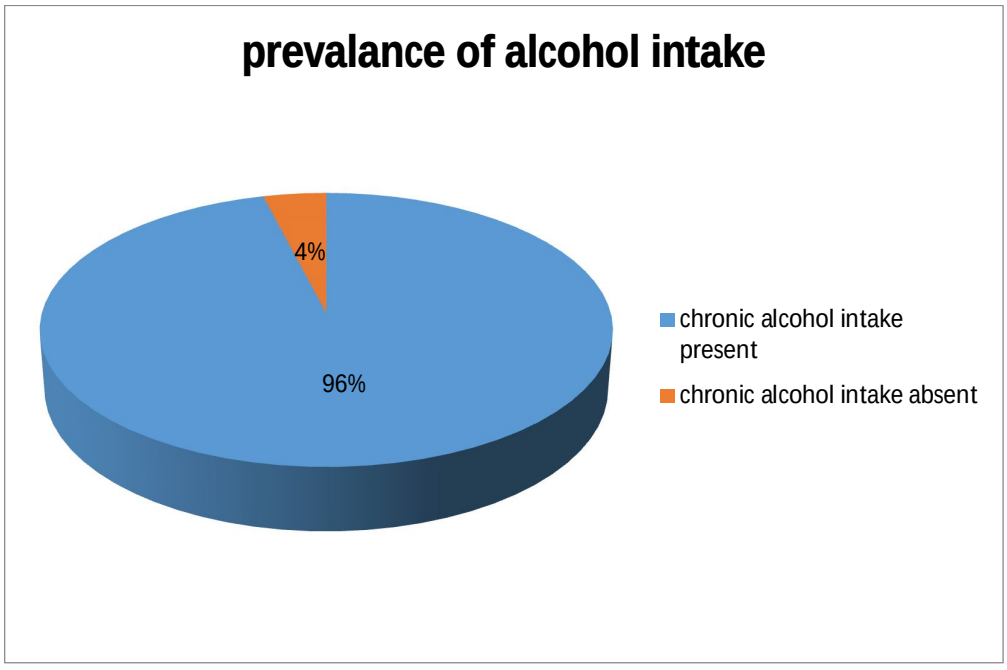
In our study , 2 female patients have been diagnosed to have acute pancreatitis due to the intake of alcohol.

In our study , the incidence of idiopathic causes of acute pancreatitis in female patients was nil.

Table 6 – History of Alcohol Intake

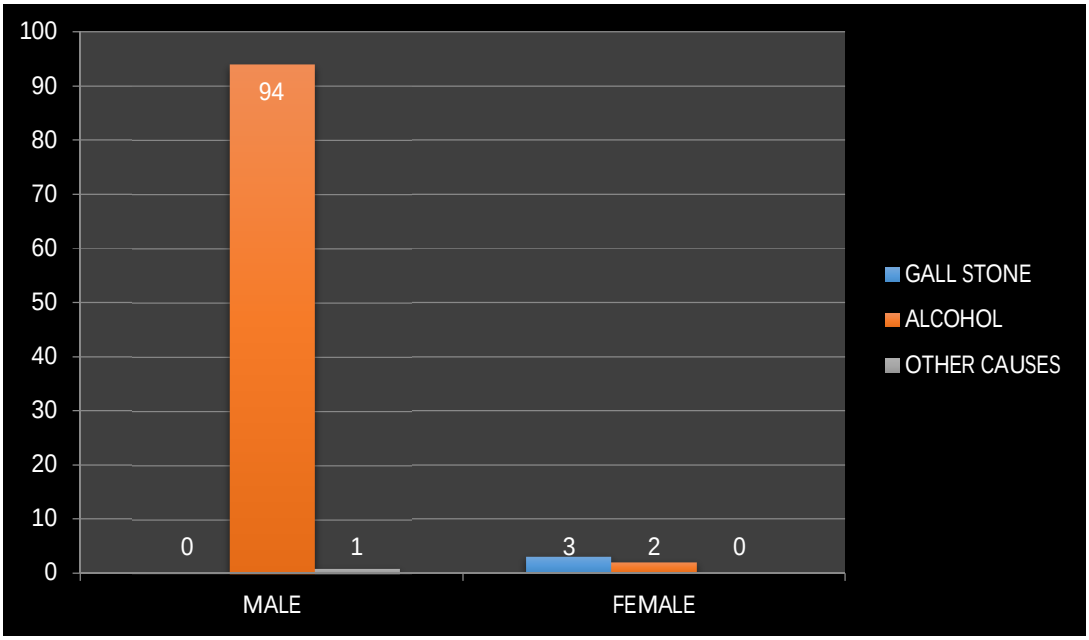
Alcohol Intake	Percentage of Study population
Present	96%
Absent	4%

From the above tabular column it is shown that 96% of the study population has a history of intake of chronic alcohol and 4% does not have a history of intake of alcohol. 96% of the study population has shown that alcohol is implicated as the causative factor of Acute pancreatitis.



96% of the study population has shown that alcohol is implicated as the causative factor of Acute pancreatitis.

COMPARISON OF MALE AND FEMALE DISTRIBUTION OF CAUSES:



The most common cause for acute pancreatitis in male is chronic alcohol intake. Out of 95 male patients with acute pancreatitis in our study 94 patients had history of chronic alcohol intake.

Out of the 5 female patients in our study 3 patients had gall stones and 2 had history of alcohol intake. On linking the etiology with the sex

Alcoholic pancreatitis is common in males

Gall stone pancreatitis is common in females.

Table 7 – Severity of Acute pancreatitis - Male:

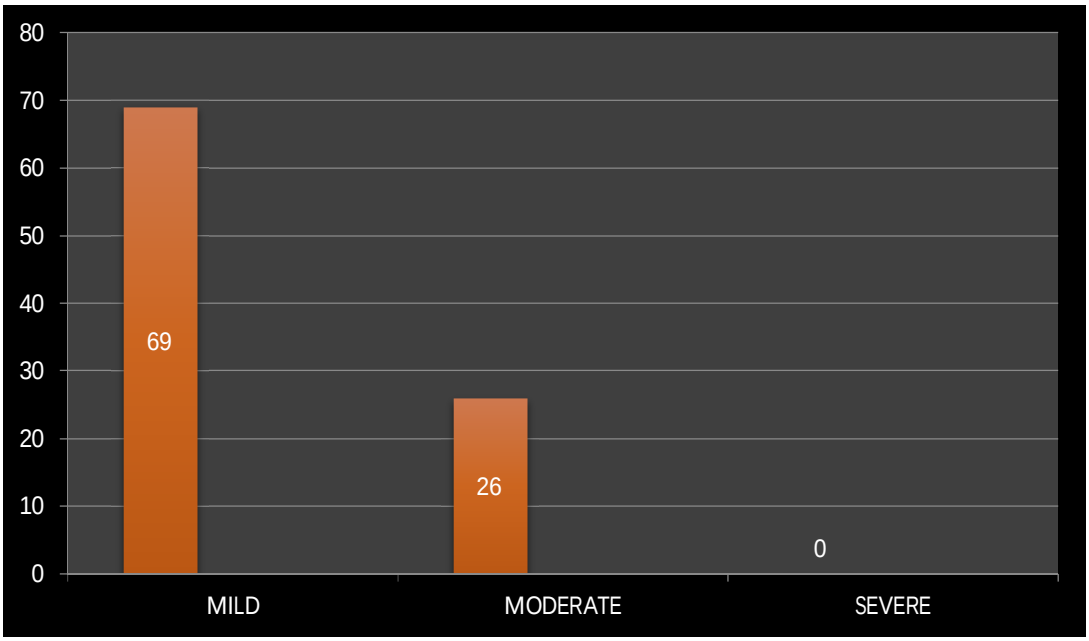
Severity	Number of patients	Percentage of Study population
Mild	69	73%
Moderate	26	27%
Severe	0	0%

From the above tabular column it is shown that 73% of the study population has severity graded as Mild according to CT severity Index.

27% of the study population has severity graded as Moderate.

0% of the study population has severity graded as Severe.

SEVERITY OF ACUTE PANCREATITIS - MALE :



From the above bar graph it is shown that 69 male patients of the study population has severity graded as Mild according to CT severity Index.

26 male patients of the study population has severity graded as Moderate.

0 male patients of the study population has severity graded as Severe.

Table 8 – Severity of Acute Pancreatitis – Female:

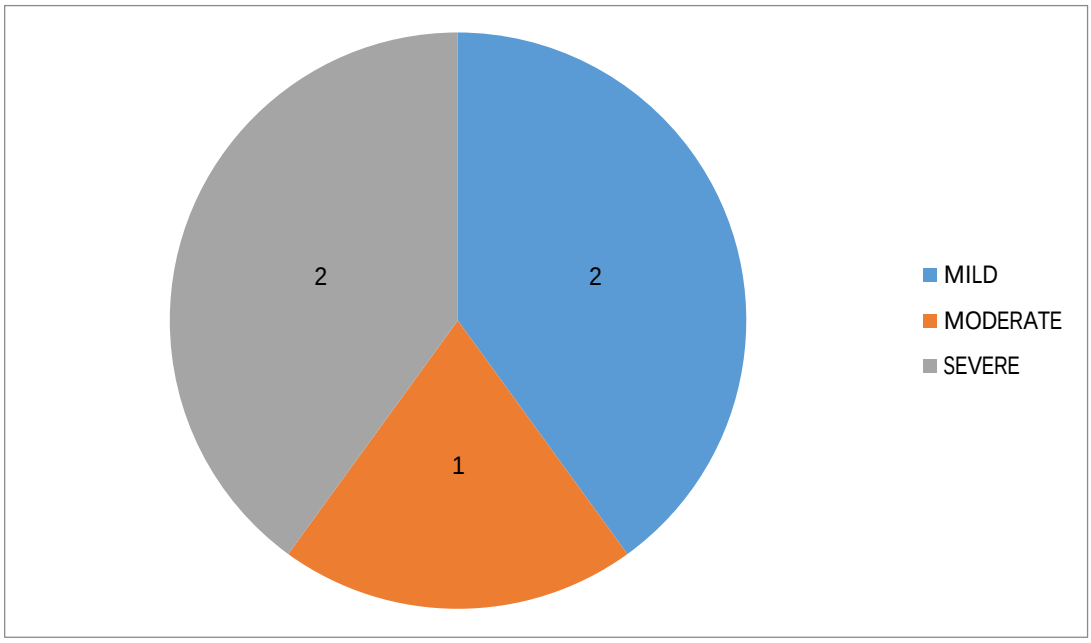
Severity	Number of patients	Percentage of Study population
Mild	2	40%
Moderate	1	20%
Severe	2	40%

From the above tabular column it is shown that 40% of the females in the study population have a severity graded as Mild according to CT severity Index.

20% of the females are graded as Moderate.

40% of the female patients were graded into Severe category.

SEVERITY IN FEMALE PATIENTS

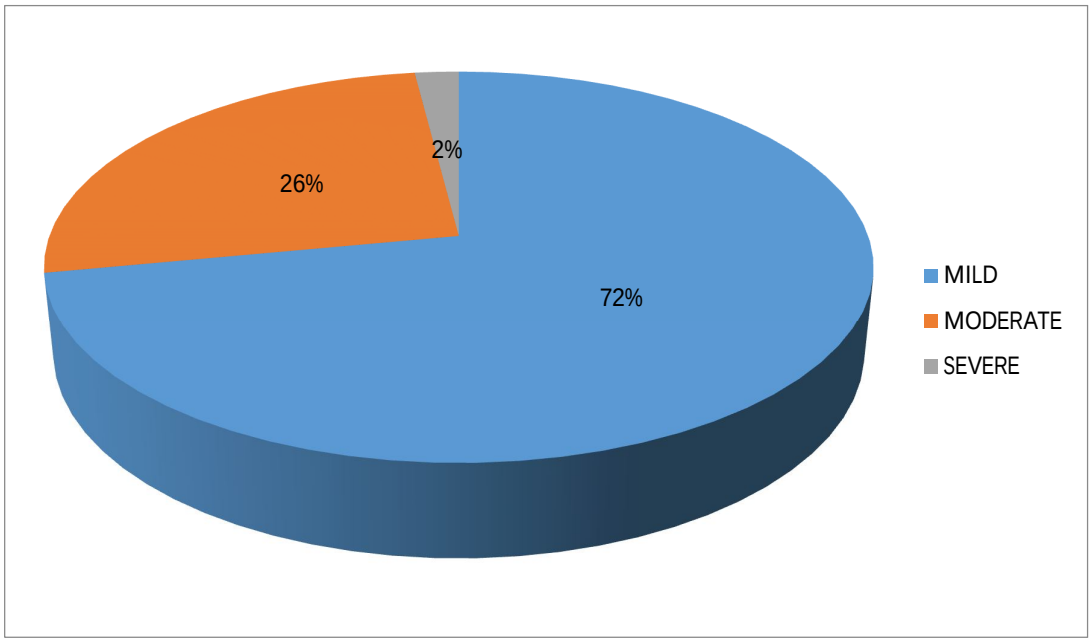


From the above pie diagram it is shown that 2 of the females in the study population have a severity graded as Mild according to CT severity Index.

1 of the females are graded as Moderate.

2 of the female patients were graded into Severe category.

SEVERITY OF ACUTE PANCREATITIS :



72% of the study population were divided into mild grade of Acute pancreatitis by the use of CT severity index.

26% of the study population were classified into moderate grade and 2% of the population were classified into severe grade.

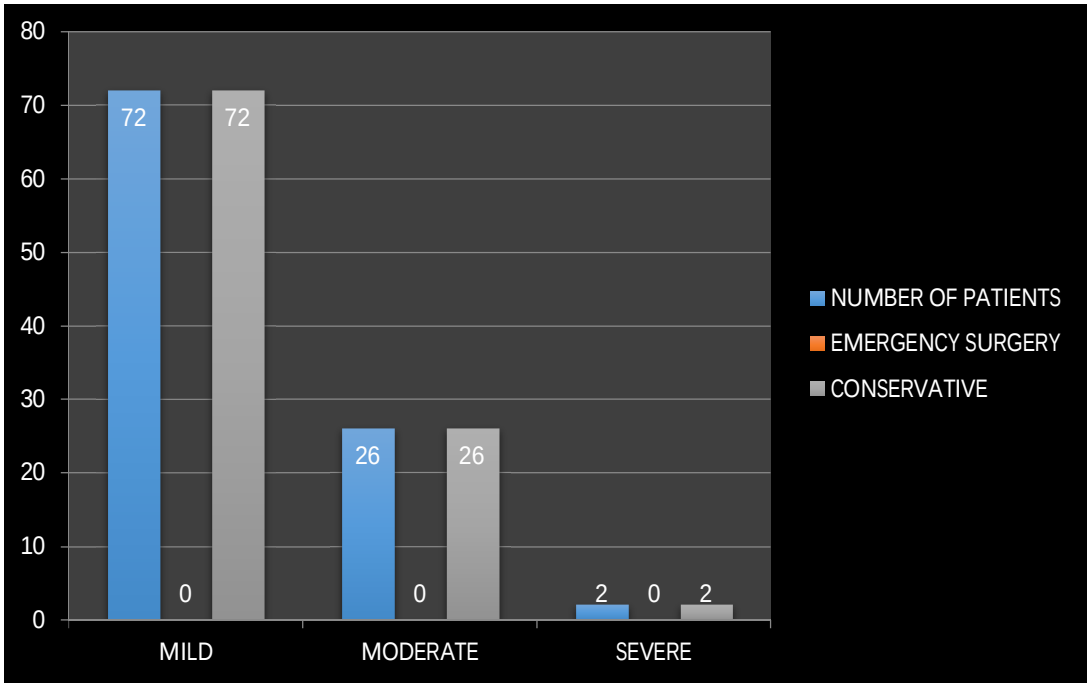
Table 9 – Management of Acute Pancreatitis :

Management	Number of patients	Percentage of Study population
Surgery	0	0%
Conservative	100	100%

Conservative management was the treatment option for all grades of Acute pancreatitis patients and surgical intervention was not performed in the study population.

All cases can be managed conservatively and does not require emergency surgical management for resolution of acute pancreatitis.

COMPARISION OF SEVERITY AND ITS MANAGEMENT FOR ACUTE PANCREATITIS :



According to this graph ,

72% of the study population were divided into mild grade of Acute pancreatitis by the use of CT severity index.

26% of the study population were classified into moderate grade and

2% of the population were classified into severe grade.

Conservative management was the treatment option for all grades of Acute pancreatitis patients and surgical intervention was not performed in the study population.

DISCUSSION

In this study , Out of 100 patients of the study population 95 patients were found to be male patients and 5 patients were female patients. This shows the acute pancreatitis is common in Male patients in comparison to female patients.

The age group in which the onset of acute pancreatitis is more common is between 31-40 which is 31% of the study population in both male and female patients of the study population. The second most common age group affected by acute pancreatitis is 41-50 age group in males and in female population we have found to have similar incidence in both 41-50 age group and more than 60 years of age group population.

The mean age of population affected by Acute pancreatitis is 39 for both males and female patients in the study population. The range of the age group affected in male patients is 20 – 66 and for female patients is 36 – 63. This indicates that the patients commonly affected by acute pancreatitis usually fall into the age group of more than 20 years and an age less than 66 years.

The most common cause of acute pancreatitis in male patients of the study population in this study is found to be chronic intake of alcohol which is 94% of the study population and 1 % of male patients were found to have acute pancreatitis due to other causes. This shows that , the most common cause of acute pancreatitis in male patients was found to excessive intake of alcohol from this study. Gall stones were not found to be the cause of acute pancreatitis in male patients of the study population and hence in this study it is shown that gall stones are the least common cause of acute pancreatitis in male patients.

From this study , it is found that the most common cause of acute pancreatitis in the female patients of the study population is gall stones which is 3 % of the study population and the second most common cause of acute pancreatitis in female patients was alcohol which is 2% of the study population and pancreatitis due to other causes was not reported in this study in female patients of the study population.

The severity graded by CT severity index of acute pancreatitis in male patients was mild , moderate and severe. 69 male patients were graded as mild grade which was 73% of the study population. 26 male patients were graded as moderate grade which was 27% of the study population. Nil patients were graded as severe by the CT severity index.

This shows that in the study population , the most prevalent grade of acute pancreatitis was mild grade which has minimal complications and can be managed conservatively. It has a good prognosis and better outcome with medical management alone and does not need any surgical intervention.

The severity in female patients was found out to be as , Mild grade in 2 female patients which was 40% of the study population. Moderate grade was found in 1 female patients which was 20% of the study population. Severe grade was found in 2 patients which was 40% of the study population. This shows that the most prevalent grade in females of the study population is mild and severe acute pancreatitis. Severe acute pancreatitis is most commonly caused by gall stones and is treated with surgical management and has good prognosis.

The management of acute pancreatitis is divided into two categories : medical and surgical management. In this study , mild grade of acute pancreatitis was managed conservatively with medical management such as intravenous fluids , intravenous antibiotics , parenteral analgesics and intravenous octreotide. Moderate grade and severe of acute pancreatitis patients were also managed conservatively with the similar medical management and found to have symptomatic

improvement with resolution of symptoms after the completion of the conservative management. Hence , this study shows that the management of both moderate and severe grades of acute pancreatitis can be done with medical management and does not require surgical management for resolution of acute pancreatitis.

CONCLUSION

To conclude, Acute pancreatitis has become a common entity in the day to day practice of general surgeons and it has become vital to evaluate the underlying causes leading to the onset of the disease and to properly categorize the patients according to the severity of the disease and accordingly manage the patients to prevent morbidity and mortality caused by the disease.

By this study , we have concluded the following :

1. Acute pancreatitis is more prevalent in the male population.

2. Acute pancreatitis most commonly involves the age group of 31- 40 years of age.

3. The most common cause leading to the onset of acute pancreatitis is chronic intake of alcohol in the male population and gall stones as a cause of acute pancreatitis has become rare due to its early detection and treatment.

4. According to the CT severity index grading system , the most common grade of acute pancreatitis is found to be mild grade and segmental involvement which has an overall good prognosis.

5. Conservative management is the treatment of choice of patients presenting with Acute pancreatitis in this study which causes an overall decrease in morbidity and mortality of the patients.

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PROFORMA FOR ACUTE PANCREATITIS

NAME: AGE: SEX: PHONE NO:

D.O.A:

ADDRESS:

COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

HISTORY OF PAST ILLNESS:

GENERAL EXAMINATION:

ABDOMINAL EXAMINATION:

1. Inspection
2. Palpation
3. Percussion
4. Auscultation

BIOCHEMICAL INVESTIGATIONS:

1. **SERUM AMYLASE**
2. **CT SEVERITY INDEX**

No of years of Alcohol Intake :

No of episodes of Abdomin pain :

Serum amylase :

MASTER CHART

S.NO	NAME	AGE	SEX	IP NO	DIAGNOSIS	AMYLASE	YrsOF ALCOHOL INTAKE	SEVERITY
1	ANANDHAVEL	36	M	33208	ACUTE ON CHRONIC PANCREATITIS	232	10	MILD
2	PONNUSAMY	51	M	26065	ACUTE ALCOHOLIC PANCREATITIS	1573	30	MILD
3	ANANDHARAJ	32	M	25125	ACUTE ALCOHOLIC PANCREATITIS	523	4	MILD
4	JOHN	40	M	21060	SEVERE ACUTE PANCREATITIS	876	12	MILD
5	KARTHIKEYAN	28	M	31785	ACUTE ALCOHOLIC PANCREATITIS	2648	4	MILD
6	MUTHUKUMAR	50	M	31158	ACUTE ALCOHOLIC PANCREATITIS	1699	22	MILD
7	CHANDRAN	28	M	22379	ACUTE PANCREATITIS WITH	332	3	MILD
8	CHANDRASEKAR	55	M	20870	PSEUDOCYST OF PANCREAS	289	25	MILD
9	ANANTH	32	M	35280	ACUTE ALCOHOLIC PANCREATITIS	651	5	MILD
10	ELANGOVAN	38	M	34852	ACUTE NECROTISING PANCREATITIS	55	10	MILD
11	PRADHAP	20	M	33135	ACUTE ALCOHOLIC PANCREATITIS	838	2	MODERATE
12	VADIVEL	23	M	67892	CHRONIC CALCIFIC PANCREATITIS	51	4	MILD
13	NATARAJAN	45	M	22838	ACUTE PANCREATITIS/ CHOLELITHIASIS	102	20	MILD
14	PETER	40	M	20430	ACUTE ALCOHOLIC PANCREATITIS	711	12	MILD

15	MURUGESAN	40	M	13165	ACUTE ALCOHOLIC PANCREATITIS	63	9	MILD
16	BASEER	49	M	9190	CHRONIC CALCIFIC PANCREATITIS	196	12	MILD
17	INAYATHULLA	52	M	5196	CHRONIC CALCIFIC PANCREATITIS	372	20	MODERATE
18	PATTU	34	M	19428	ACUTE ALCOHOLIC PANCREATITIS	71	10	MODERATE
19	CHANDRASEKAR	55	M	35865	ACUTE ALCOHOLIC PANCREATITIS	894	15	MILD
20	SENTHIL	34	M	59190	ACUTE ALCOHOLIC PANCREATITIS	658	12	MILD
21	GANESAN	45	M	60001	ACUTE ALCOHOLIC PANCREATITIS	159	10	MILD
22	VIJAYAPANDIAN	27	M	49892	ACUTE ALCOHOLIC PANCREATITIS	144	5	MILD
23	ABUSALIYA	27	M	60013	ACUTE ALCOHOLIC PANCREATITIS	874	7	MODERATE
24	DOMINIK	51	M	60622	ACUTE ALCOHOLIC PANCREATITIS	211	8	MODERATE
25	DAVID	22	M	41640	ACUTE ALCOHOLIC PANCREATITIS	658	4	MODERATE
26	CHANDRAN	28	M	39597	ACUTE ALCOHOLIC PANCREATITIS	385	3	MILD
27	PRABAKARAN	31	M	41811	ACUTE ALCOHOLIC PANCREATITIS	825	6	MILD
28	RAJI	33	M	43832	CHRONIC CALCIFIC PANCREATITIS	115	10	MILD
29	MANICKAM	30	M	10813	ACUTE ALCOHOLIC PANCREATITIS	259	8	MILD
30	RAJADURAI	18	M	6113	ACUTE PANCREATITIS WITH GB SLUDGE	214	2	MILD
31	SENTHILKUMAR	33	M	1759	ACUTE ALCOHOLIC PANCREATITIS	406	4	MILD
32	SUNDAR RAJ	49	M	3864	ACUTE PANCREATITIS WITH FOCAL			
33	MAHESH	24	M	3916	ACUTE PANCREATITIS	308	2 YEARS	MILD

34	RANGANATHAN	38	M	6012	ACUTE NECROTISING PANCREATITIS	1076	10	MILD
35	ARJUN	42	M	42390	ACUTE ON CHRONIC PANCREATITIS	466	12	MILD
36	SRINIVASAN	42	M	33207	ACUTE ON CHRONIC PANCREATITIS	302	7	MODERATE
37	SARAVANAKUMAR	36	M	40428	ACUTE ON CHRONIC PANCREATITIS	409	8	MILD
38	NAGARAJ	45	M	40285	ACUTE PANCREATITIS	504	5	MILD
39	RASU	35	M	41292	ACUTE PANCREATITIS	306	7	MILD
40	SARAVANAKUMAR	30	M	58804	ACUTE PANCREATITIS	298	10	MILD
41	MARUTHACHALAM	44	M	36940	ACUTE PANCREATITIS	426	12	MODERATE
42	MANIKANDAN	40	M	59486	ACUTE PANCREATITIS	304	8	MILD
43	ABDHUL RAHEEM	37	M	60834	ACUTE PANCREATITIS	514	5	MILD
44	MOORTHY	32	M	60828	ACUTE PANCREATITIS	408	2	MODERATE
45	MANICKKAM	55	M	61084	ACUTE PANCREATITIS	640	11	MILD
46	ALLIMUTHU	55	M	61666	ACUTE PANCREATITIS	1204	12	MILD
47	BAVANKUMAR	17	M	61746	ACUTE PANCREATITIS/ PANCREATIC DUCT CALCULI	502		MILD
48	NAVEEN BHARATHI	48	M	62733	ACUTE ALCOHOLIC PANREATITIS			
49	MANICKAM	40	M	62920	ACUTE ALCOHOLIC PANCREATITIS	436	6	MILD
50	RAGHUPATHI	54	M	62733	ACUTE ALCOHOLIC PANCREATITIS	304	9	MODERATE

	BABU							
51	MAHENDRAN	29	M	62920	ACUTE ALCOHOLIC PANCREATITIS	283	3	MODERATE
52	ARUN PRAKASH	35	M	61400	ACUTE ALCOHOLIC PANCREATITIS	306	5	MODERATE
53	SELVA NAYAGAM	57	M	63329	SEVERE ACUTE PANCREATITIS	152	13	MILD
54	NAGARAJ	44	M	63523	ACUTE SEVERE ALCOHOLIC PANCREATITIS	747	20	MILD
55	JEYAKUMAR	30	M	63892	ACUTE SEVERE PANCREATITIS	457	10	MODERATE
56	RAMU	20	M	73866	ACUTE SEVERE PANCREATITIS	917	3	MODERATE
57	ANANDA KUMAR	30	M	67043	ACUTE ON CHRONIC PANCREATITIS	642	15	MILD
58	KUMAR	39	M	64827	ACUTE ALCOHOLIC PANCREATITITS	925	15	MILD
59	FATHIMA	63	M	59834	GALL STONE PANCREATITIS	158	NIL	MILD
60	NAYAK	25	M	59060	ACUTEV ALCOHOLIC PANCREATITIS	2738	NIL	MILD
61	USSAIN BABU	37	M	70798	ACUTE ON CHRONIC PANCREATITIS	1691	15	MODERATE
62	SUDISH	44	M	58157	ACUTE ON CHRONIC PANCREATITIS	3082	20	MILD
63	KARTHIK	24	M	66373	ACUTE ALCOHOLIC PANCREATITIS	2037	6	MILD
64	MAHALINGAM	48	M	65078	ACUTE ALCOHOLIC PANCREATITIS	2855	20	MODERATE
65	ARJUNAN	42	M	60008	ACUTE ALCOHOLIC PANCREATITIS	1275	20	MODERATE
66	SURESH	35	M	58753	ACUTE ALCOHOLIC PANCREATITIS	571	10	MODERATE
67	SIVASAMY	40	M	71454	ACUTE ALCOHOLIC PANCREATITIS	1616	10	MILD

68	GUNA	20	M	71341	ACUTE ALCOHOLIC PANCREATITIS	336	4	MODERATE
69	PETER	42	M	53912	ACUTE ON CHRONIC PANCREATITIS	152	20	MILD
70	SELVARAJ	52	M	16007	ACUTE ALCOHOLIC PANCREATITIS	476	34	MILD
71	ANANDHAKUMAR	26	M	48884	ACUTE ALCOHOLIC PANCREATITIS	1322	10	MILD
72	NACHIMUTHU	40	M	47612	ACUTE NECROTISING PANCREATITIS	2871	20	MILD
73	VIJAY	37	M	46250	ACUTE ALCOHOLIC PANCREATITIS	561	16	MILD
74	MATHEWS	63	M	44122	ACUTE ALCOHOLIC PANCREATITIS	486	26	MILD
75	KULANTHAISAMY	43	M	69416	GALL STONE PANCREATITIS	324	14	MODERATE
76	PRAKASH	45	M	50522	ACUTE ALCOHOLIC PANCREATITIS	410	18	MODERATE
77	SENTHIL KUMAR	54	M	66706	ACUTE ALCOHOLIC PANCREATITIS	510	25	MODERATE
78	MURUGESAN	38	M	52111	GALL STONE PANCREATITIS	210	14	MODERATE
79	BANNARI	40	F	48914	GALL STONE PANCREATITIS	160	NIL	SEVERE
80	SENTHIL KUMAR	42	M	47322	ACUTE ALCOHOLIC PANCREATITIS	800	18	MILD
81	MOHAMAD	46	M	71470	ACUTE ALCOHOLIC PANCREATITIS	960	22	MILD
82	RAMESH	52	M	33787	ACUTE ALCOHOLIC PANCREATITIS	1040	24	MILD
83	RAJAN	56	M	33800	ACUTE ALCOHOLIC PANCREATITIS	619	24	MODERATE
84	KANNAN	48	M	36916	ACUTE ALCOHOLIC PANCREATITIS	560	22	MILD
85	DURAIRAJ	50	M	38342	ACUTE ALCOHOLIC PANCREATITIS	480	25	MILD
86	SHANTHI	36	F	38459	GALL STONE PANCREATITIS	260	NIL	SEVERE

87	MOORTHY	46	M	40252	ACUTE ALCOHOLIC PANCREATITIS	800	21	MILD
88	SELVARAJ	66	M	41360	ACUTE ALCOHOLIC PANCREATITIS	1640	36	MILD
89	CHELLAM	48	F	42799	GALL STONE PANCREATITIS	320	NIL	MILD
90	NARAYANAN	53	M	45075	ACUTE ALCOHOLIC PANCREATITIS	1420	25	MILD
91	POONKODI	49	M	52930	GALL STONE PANCREATITIS	210	NIL	MILD
92	SIVAKUMAR	46	M	60361	ACUTE ALCOHOLIC PANCREATITIS	960	22	MILD
93	RANI	43	F	58934	GALL STONE PANCREATITIS	200	NIL	MODERATE
94	BALAMURUGAN	40	M	71987	IDIOPATHIC PANCREARTITIS	610	NIL	MODERATE
95	PONNAIYAN	54	M	70812	ACUTE ALCOHOLIC PANCREATITIS	430	24	MODERATE
96	CHINNAN	58	M	61754	ACUTE ALCOHOLIC PANCREATITIS	1600	30	MILD
97	BALA KUMAR	54	M	54298	ACUTE ALCOHOLIC PANCREATITIS	1430	30	MILD
98	JEGADEESH	52	M	39524	ACUTE ALCOHOLIC PANCREATITIS	1200	22	MILD
99	ARUKUTTY	55	M	17552	ACUTE ALCOHOLIC PANCREATITIS	1160	29	MILD
100	ARUSAMY	66	M	16797	ACUTE ALCOHOLIC PANCREATITIS	1200	40	MILD